THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—21ST YEAR. SYDNEY, SATURDAY, MAY 12, 1934. No. 19.

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THE INTERPRETATION OF SYSTOLIC MURMURS.[1]

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IT is my intention this evening to discuss the commonest abnormal physical sign found on auscultation of the heart, that is, a systolic murmur heard at or near the apex beat of the heart; and I propose to show the varying inferences which have been drawn from this sign and compare them with the views now held.

In order to do this the question of whether a dramatic change has occurred in the type of mitral disease now seen must be ventilated, and I hope that a discussion on this particular point will ensue as a result of my rather dogmatic statements; for it would seem that the best way to produce a free discussion at these meetings is to express a definite, even dogmatic, view, which can either be supported or opposed by those interested in the subject.

All history of disorders of circulation of the blood must date from the discoverer of the circulation, William Harvey, but both forms of mitral disease, obstructive and regurgitant, were in all probability first recognized by Morgagni (1682-1771). The clinical recognition, however, is usually ascribed to Laennec (1781-1826); but as Laennec believed that the second sound of the heart was due to auricular systole, it is not to be wondered that his work, as read in Forbes's translation, is most obscure; it is improbable that he was able to distinguish between the two forms by auscultation alone. Although Hope in 1832 wrote an article on the signs and symptoms of mitral disease, his description of the sounds produced by obstructive murmurs would not fit in with modern observations, and Fauvel in 1843 appears to have been the first

[1] Read at a meeting of the South Australian Branch of the British Medical Association on November 30, 1933.

clinician who could recognize the two disorders with any certainty.

The next forward step was taken by Balfour in 1876, who maintained that organic disease of the mitral leaflets sufficient to produce insufficiency was always accompanied with a certain degree of narrowing of the orifice, so that the only unequivocal clinical proof of actual disease of the mitral valve was a presystolic murmur. Though this view was brought forward with increasing frequency and insistency, nearer 50 than 40 years elapsed before it found favour with the writers of text books; even Osler was cautious, possibly because this section of his last text book was written by A. G. Gibson; it contains the astounding statement: "With an apex beat in the usual situation and a regular rhythm the auscultatory phenomena may be practically disregarded", thus ignoring the potentialities of a presystolic mitral murmur, which usually is accompanied by a normally placed apex beat.

Sansom and A. G. Gibson (1898) state:

> The chief sign by which the diagnosis of mitral regurgitation is made is an apical systolic murmur; this abnormal sound is often musical, and is heard over the whole precordium, often conducted to the left axilla and beyond to the angle of the scapula.

This statement is taken from Allbutt and Rolleston's "System of Medicine", and reflects the opinion of the vast majority of the leading clinicians of the time, the only doubting voice being that of Balfour (1876), who did not believe that there was any true valvular incompetence without obstruction.

A. G. Gibson, in "Osler's Modern Medicine" (1921), writes that a murmur, systolic in time and of maximum intensity at the apex, and propagated even to the axilla, does not necessarily indicate mitral insufficiency. Osler, with his unrivalled combination of pathological and clinical experience, casts a doubt on the certainty that a pathological basis always underlies the classical sign.

East and Bain, in "Recent Advances in Cardiology" (1929), write that a harsh or musical murmur, far conducted, at the apex, shows permanent damage and suggests the development of mitral stenosis. They definitely withdraw the classical sign from regurgitation and hand it over unhesitatingly to obstruction.

In order to examine this problem in a dispassionate way, let us first consider what are the causes of mitral regurgitation from a mechanical point of view, and secondly, what are the *post mortem* evidences that mitral regurgitation has existed in any given case?

Insufficiency of the mitral valve is due in the vast majority of cases to two different conditions, though possibly the two conditions may coexist: (i) Changes in the valve segments, whereby they are shortened and contracted, associated with changes in the *chordæ tendineæ*; (ii) changes in the muscular wall of the ventricle, either dilatation, so that the valve segments fail to close an enlarged orifice, or infarction, so that the basal support of either the valve segments or the papillary muscles is impaired.

If we examine the first causative condition, we see at once that the stiffening of the valve segment, which is sufficient to cause regurgitation, will at the same time cause obstruction, as it is due to a shortening of the valve segment. Hence pure regurgitation cannot result from pure leaflet deformation; for even in the more florid forms of endocarditis a vegetation large enough and sessile enough to hold the segments apart would probably cause some degree of obstruction. Therefore, in the valvular causes of pure regurgitation we are limited to disease of the *chordæ tendineæ*, and I can find no evidence that these structures are ever affected without concurrent valvular lesions, nor is it likely, seeing that the essential lesion is now thought to be an endocardial nodule, found in the early stages in the valves and left auricular wall (Shaw and McCallum), the subsequent fibrosis of the former leading to valvular deformity. However, Osler describes a form of puckering and curling of the valve segments, associated with lengthened *chordæ tendineæ*, which he believed could cause a pure mitral regurgitation in children.

In the second condition cases of dilatation of the ventricle to such an extent that the mitral valve segments could not coapt are recorded, but are extremely rare, and I think it unlikely that, except in very few cases of poisoning of the muscle of the ventricular wall by toxic products, such as occurs in diphtheria and pneumonia, the dilatation is sufficiently grave to permit of regurgitation through the mitral valve. However, in infarction, especially when the papillary muscles are included in the ischæmic area, their enfeeblement may give rise to a failure of accurate contact of the valve segments and the instant development of a valvular insufficiency.

One very real difficulty in the way of correlating certain murmurs with mitral regurgitation lies in the frequent impossibility of determining, after death, from the appearance of the cusps, whether leakage has occurred during life. Fifty-five years ago Duroziez declared that if the orifice was large enough to admit the thumb regurgitation had occurred, otherwise the signs would have been those of obstruction. If we consider that group of cases in which regurgitation is certain to have occurred, that is, when valves are so rigid as to be incapable of opening further or closing further, cases now classed as stenosis irrespective of Duroziez's dictum, we may well ask how many gave the sign of regurgitation during life (systolic murmur). In fully one-third of them there has been no systolic murmur at all; in the remaining two-thirds the murmur has had very variable characteristics.

Turning now to the clinical aspect, let us consider the variety of systolic apical murmurs. These are generally placed in four or more groups.

1. Constant murmurs, that is, constant from cycle to cycle and constant from day to day. They

are in general harsher and longer murmurs, and are heard over the precordium and out to the axilla. These are the murmurs which are chiefly regarded as signifying mitral regurgitation.

2. Inconstant murmurs, soft and blowing in character, and often heard in one posture only, and that most often the recumbent. These murmurs vary from time to time, they vary with exercise, and are usually only audible over a localized apical area.

3. Exocardial murmurs — precordial murmurs that are short, superficial and with a scraping quality. They are thought to be due to a rough precordium or pleura. On those uncommon occasions when an opportunity offers of examining the suspected membrane, proof of its disease is rare, that is, excepting the characteristic findings of recent precarditis.

4. Cardio-respiratory murmurs. These are the commonest apical murmurs; but they may extend to the scapula or left axilla. They are produced by the breaking of the normal vesicular murmur of inspiration into two or more short murmurs by the ventricular systoles. The ventricle during systole creates a little vacuum and accelerates the rate at which air enters the lung; consequently this murmur is a normal phenomenon during exercise or excitement.

Mitral Regurgitation and Its Sign.

There are no symptoms which can be attributed to mitral regurgitation, and the only sign is a systolic murmur. It is frequently said that this murmur replaces the first cardiac sound; graphic records always show the first sound to be present. Whereas the systolic murmur, which, though subject to considerable variation, shows the characteristics of those in the first group above (constant murmurs), is usually attributed to regurgitation, yet every variety of systolic murmur may be found in cases which at *post mortem* examination show no real damage to the valves. In these cases it is usual to suppose that the muscular ring surrounding the base of the valve is sufficiently relaxed to cause leakage.

It is never very satisfactory to base a diagnosis on a single sign, and less so when the sign is known to have only a moderate degree of reliability. If we are treating a patient with acute infection, or a frail, elderly person, and we are sure that a systolic murmur has only recently developed near the impulse, it is possible that the heart is dilating; but this conclusion should never be definite unless at the same time, or soon afterwards, direct signs of enlargement can be demonstrated.

Confidence in the value of a diagnosis of mitral regurgitation is therefore reduced both by doubt as to its very existence and by our inability to estimate the degree. Consequently the diagnosis of mitral regurgitation has a very limited importance and is probably most useful (i) in acute infectious disease as an early indication of muscular failure, or (ii) in the muscular damage caused by infarction.

To recognize mitral disease by means of the systolic murmur it is first essential to eliminate all murmurs which do not arise from the mitral valve and next to distinguish between the murmur of a diseased ventricle and a diseased valve. The strongest statement that can be defended is "that a long, harsh systolic murmur, heard beyond the impulse in a person whose past history discloses rheumatic fever, signifies mitral disease with regurgitation", and there are many who would attack the second element, namely, regurgitation, and claim that diagnosis of mitral disease depends solely on the recognition of obstruction.

How, then, has this altered state of affairs come about? Is it (i) that the type of mitral disease now encountered is different from that seen sixty years ago, or (ii) that the grave prognostic significance of fibrillation and its response to treatment have focused our attention on stenosis, or (iii) that new diagnostic methods have supplanted the older ones? It would seem that (i) is probable, (ii) is possible, and (iii) unlikely.

The authors of text books are always liable to repeat certain statements from one edition to another, long after general medical opinion on the point has altered, and probably this conservatism has a sound basis. One finds that the statement that mitral regurgitation is the commonest variety of valvular disease is fighting an excellent rear-guard action. Dr. H. Swift and Dr. A. A. Lendon assure me that in their student days mitral stenosis was infinitely rarer than mitral regurgitation, whereas nowadays it is hard to find anyone brave enough to diagnose the latter condition. If new instrumental methods of diagnosis have any real bearing on the question, then perhaps we should have a ready explanation of the problem; but the distinction between the two is still made by palpation and auscultation, arts which have declined rather than advanced in our time.

The correlation of cause and effect was as follows. If a patient had a single attack of rheumatic fever and valvulitis developed, then regurgitation would be the result; if repeated attacks of rheumatic fever, then "double mitral" disease would follow; but if there were no history of fever, but only of vague or mild rheumatic manifestations, then pure mitral stenosis would ensue.

Even in the last twenty years we have seen the rapid disappearance of the "florid" rheumatic fever cases; like chlorosis, rheumatic fever has almost disappeared, at any rate from amongst those persons eligible by age or otherwise for admission to the Adelaide Hospital. This, then, would be the reason I would give for the disappearance of mitral regurgitation: the disappearance of its cause. I think that this factor largely outweighs the idea of an alteration in diagnostic fashion.

With the advent of compulsory military service in Great Britain the practice of passing as fit recruits with systolic murmurs, normal rhythm and absence of cardiac enlargement became general, and

these men were able to stand, without ill effects, the arduous demands of active service. Lewis gives an outstanding example of exercise tolerance in a case of this type; he states that the individual who climbed higher up the slopes of Mount Everest than any living man has a loud apical systolic murmur, transmitted to his axilla, which is surely an excellent confirmation of the principles outlined above.

Another systolic murmur, also heard toward the lower portion of the heart, the murmur of tricuspid regurgitation, may be used to exemplify another change of front in cardiological opinion. This murmur, which is by no means uncommon in venous congestion, suffers from much the same disabilities as its corresponding mitral regurgitant murmur; for the diagnosis in general is simply the discovery of a murmur, which is assumed, possibly correctly, to indicate this backward flow. The degree of influence of this reflux cannot be estimated, except from abnormal waves in the cervical veins, and this is doubtful.

Cardiac Failure.

The back pressure theory of cardiac failure postulated that as a result of a valve lesion a chamber of the heart dilated when it was unable to do the extra work required of it. If no valve lesion existed, then a functional valvular inefficiency might be caused by a primary dilatation. The ensuing regurgitation resulted in a damming up of the blood behind the chamber first involved and gradually led to the signs of heart failure.

The *vis a tergo* theory postulated that the heart failed as a whole and the phenomena of œdema and venous congestion were ascribed rather to a stagnation of the blood stream than to any back pressure. The pulmonary congestion, consequently, was due to right ventricular inefficiency, and venous engorgement and œdema to failure of the left ventricle. Obviously this theory was diametrically opposed to its predecessor.

The French authors have always maintained that primary failure of one ventricle could and should be distinguished from failure of the other, and the terms *asystole gauche* and *asystole droite* are freely used. Some writers even recommend different drugs for each type of failure.

With regard to the back pressure theory, the sequence of mitral incompetence and failure would be: regurgitation to the left auricle, congestion of the pulmonary circulation, dilatation of the right ventricle, tricuspid regurgitation, engorgement of the veins and liver, dropsy.

Mackenzie was a determined opponent of the back pressure idea, largely because of the ineffectiveness of prognosis based solely on valvular defects. He found that "decompensation", as it was termed, depended more on muscular inefficiency, as exemplified by cardiac enlargement and auricular fibrillation, and he introduced the generic term "heart failure" as a substitute, and was the main sponsor of the *vis a tergo* theory. In the back pressure theory the tricuspid valve became the key point of the defence. Incompetence of this valve became the signal for engorgement of the veins, and the two terms were practically synonymous. Two facts were overlooked: that by obstruction to the outlet of blood from the right ventricle this chamber cannot be engorged without a corresponding rise of venous pressure, and that venous pressure rises gradually, not abruptly, as it would in sudden tricuspid incompetence. Moreover, cardiac failure supervenes in hearts without valvular lesions or murmurs to suggest functional incompetence.

With regard to the *vis a tergo* theory, it is correct to ascribe all heart failure to inefficiency of the heart muscle, and the theory would be correct in its entirety if all failure were always a gradual process, in which ventricular contraction became weaker and weaker uninfluenced by any outside factors. Very occasionally this is true, and it is best exemplified by the failure of the hearts of old people who have taken very good care of themselves. Experimental work has shown definitely that œdema depends on a slowing of the circulation rate to one-half the normal rate, and these elderly people may have œdema without any great distress. Far more often, however, heart failure is precipitated by some sudden event, such as the onset of an abnormal rhythm or an attempt at some exertion beyond the power of the heart, which suddenly becomes inefficient and fails to deal with the blood returning to it. The fibres are stretched beyond their optimum length, dilatation ensues, and the pressure rises behind the dilated ventricle.

The back pressure theory, then, was correct in associating venous engorgement with right ventricular failure, but wrong in its explanation of the method, and the *vis a tergo* theory was right in ascribing œdema to circulation slowing from muscular inefficiency.

True acute heart failure is due either to ventricular fibrillation or massive coronary thrombosis and is inseparable from sudden death. All other failure is subacute or chronic.

Subacute heart failure is of two kinds. In the first type, prior to failure, the circulatory speed is normal, for example, rapid myocardial failure in diphtheria or pneumonia. The heart becomes rapidly unable to deal with the blood returning to it, the chambers dilate, and the blood accumulates in the venous system and liver, for the left side can only send on the blood forwarded to it by the right side. There is no œdema of the extremities, and usually there is no congestion of the lungs.

In the second type the circulatory speed prior to failure is subnormal from previous heart disease. Failure is precipitated by some sudden event, such as fibrillation; slowing of the stream below the critical level occurs and dropsy and congestion of the lung bases follow.

Finally I should like to refer again to the view of independent failure of the two sides of the heart.

It is based on the theory that mechanical interference with the work of either ventricle will retard the circulation behind the affected chamber. This means that the pulmonary circulation will become congested in left-sided failure, and when the systemic veins become engorged the right ventricle is failing.

Failure of the Left Side of the Heart.

In aortic reflux or high blood pressure the systemic blood flow maintains its velocity whilst the pulmonary velocity is slowed, with resultant congestion at the bases of the lungs. When failure of the left side of the heart is relatively acute, the result may be acute pulmonary œdema, a condition at one time associated with failure of the right side of the heart.

The pulmonary congestion which arises in association with mitral stenosis is due to retardation of the pulmonary flow, without any failure of the left side of the heart.

Failure of the Right Side of the Heart.

Primary failure of the right side of the heart is best seen in emphysema and also in pulmonary embolism; but owing to the law of coordinate contraction, blood accumulates behind the right side of the heart in any sudden heart failure.

Though engorgement of the venous system may show itself by an enlarged and pulsating liver, the most satisfactory evidence of this state is found in the distended external jugular veins, in which, as Lewis has been at great pains to emphasize, the level of the top of the blood column is raised above the right auricle, and we should estimate the difference in vertical levels and not the anatomical point reached by this blood column.

Acknowledgements.

Before terminating this summary of the conflicting ancient and modern views I should like to draw attention to the fact that much of the experimental work on the circulation which made the new views tenable was done by an Adelaide graduate, Dr. Howard Florey.

I should also like to acknowledge that many of the statements made in this paper are taken from Lewis's recent work on cardiology, and the description of back pressure and *vis a tergo* from East and Bain's "Recent Advances in Cardiology".

THE ÆTIOLOGY AND TREATMENT OF PUERPERAL AND NON-PUERPERAL PELVIC INFLAMMATION.[1]

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IT is the intention in a brief paper this evening to limit the discussion on the ætiology and treatment of pelvic inflammation to non-tuberculous inflammatory conditions within the pelvis outside the uterus, that is, pathological conditions involving the Fallopian tube, ovary, pelvic peritoneum or cellular tissue singly or more or less collectively.

[1] Read at a meeting of the Section of Gynæcology and Obstetrics of the Victorian Branch of the British Medical Association on March 13, 1934.

Ætiology.

It has been stated that practically every case of "primary" acute pelvic inflammation can be traced to infection from labour, from abortion, from instrumentation, or from gonorrhœa, and that the causative agent is either one of the pyogenic organisms of the streptococcal class or the gonococcus. The gonococcus, having a natural *penchant* for mucous and serous membranes, would seem to have an ideal habitat in the mucous lining of the vagina, uterus and tube, along which it can spread, causing devastation in its path, to involve finally the peritoneum and adjacent structures. The pyogenic organisms of the streptococcal class, however, may extend directly to the tube from the infected site in the uterus, or by way of the lymphatics or through thrombosed sinuses in the uterine wall to the surrounding connective tissue, thus giving rise to a pelvic cellulitis or parametritis with varying degrees of perisalpingitis.

Crossen has made the following statements: (i) As a general proposition it may be said that the gonococcus is the only germ that will spontaneously invade the normal non-puerperal uterus and tubes; and (ii) nearly all the streptococcic inflammatory masses in the pelvis can be traced to sepsis following labour or miscarriage, and further, that in the adult streptococci do not penetrate a non-puerperal uterus.

Accepting these statements, then, the majority of pelvic infections would appear to fall into two main clinical classes: (i) a gonococcal or non-puerperal and (ii) a streptococcal or puerperal. The history and the location of the lesion supply the indications into which classification each particular case would be placed.

Researches into the bacteriology and pathology of pelvic infection in recent years are as follows: In 634 cases of chronic suppuration in the pelvis collected by Andrews, the bacteriological findings, excluding tuberculous cases, were as follows:

Sterile	55·0%
Saprophytic	6·0%
Gonococcus	22·5%
Streptococcus and staphylococcus	12·0%
Pneumococcus	2·0%
Bacillus coli communis	2·5%

Curtis,[(1)] of Chicago, in his report dealing with the pathology and bacteriology of the Fallopian tubes in a series of 300 cases, proved that in 70% of tubes excised the inflammatory condition was definitely gonococcal; this was also suspected, but not proved, in another 10% of cases. In 15% other pus-producing organisms, such as various types of streptococci, were present. *Bacillus coli communis* and mixed microorganisms were infrequent as a primary cause of infection. In 5% evidence of tuberculosis was found, which is rather a high percentage.

Contrasting the bacteriological findings of Andrews with the bacteriological and pathological findings of Curtis, we find a marked similarity. Curtis proved that the gonococcus was the infective agent in 70% and suspected in 10% of the excised tubes in his series. Andrews demonstrated the gonococcus in 22·5% with 55% sterile; but, knowing that the gonococcus soon disappears from the uterine and tubal mucosa, one could assume that the majority of the 55% of sterile cases were of gonococcal origin. If we add these together we would get a 77% gonococcal, which is approximately the same result as that arrived at by Curtis.

Curtis is convincing on account of his far-reaching investigations, not only of the bacteriological conditions present, but the summarizing of these with the macroscopic appearances at the time of operation with the subsequent microscopic examinations. He states his findings in this wise: In the gonococcal class we find at the time of operation thickening and induration of the tube with closure of the fimbriated extremity, the pelvic adhesions being easily amenable to separation by blunt dissection. Microscopically the folds of the mucosa are found to be adherent, pockets of gland-like columnar epithelium extend deeply into the walls of the tube, blood vessels are numerous and plasma cells are characteristic.

In a streptococcal infection we find that tubal infection is usually but a part of the picture; perisalpingitis is the most frequent type of tubal lesion. Even though there be extensive salpingitis, the fimbriated extremities will very likely remain open and the mucous membrane folds or villi of such tubes show few adhesions.

Munro Kerr states that salpingitis following abortion or pregnancy takes place by different routes. In the former infection has easy access to the tube from the uterine cavity. In the latter, the development of the pregnant uterus has disturbed the course of the tube through the uterine wall in such an oblique direction that the tube is practically protected by a valve; in the puerperium, therefore, the route of infection is more commonly a lymphatic spread via the broad ligament which reaches the lumen of the tube through its wall. This view meets that of Curtis half way, namely, that the chief disturbances following an infected site in the puerperal uterus are in the nature of a parametritis and varying degrees of perisalpingitis.

Hertzler, in one of his masterly monographs, compares the more stormy, though less serious, constitutional disturbances of gonococcal salpingitis with those produced by pus microorganisms, and further contrasts the pathological changes in the tube in either class.

He states that of all the bacteria the gonococcus produces the greatest amount of œdema. With the extension of the inflammation a subperitoneal œdema forms, adding to the normal dimensions of the tube, but the œdematous neighbouring organs are attached to the tube and produce the bulk of the pelvic mass. The uterus becomes fixed, as if encased in plaster of Paris. The outcome of this may be a recession with considerable resolution of the diseased process. This may even be complete, but usually adhesions are formed and the fimbriæ are closed, so that the function of the tube is lost.

Contrast this with the results brought about in the tube when the infection is of post-abortal or puerperal origin, or the result of instrumentation, or infected wounds or of erosions of the cervix. Here the generalized sepsis is apt to overshadow the picture, and invasion of the parametrial tissue may be more in evidence than the involvement of the tubes. It is the milder infections only that play their chief rôle within the tube; material for study is usually made available either by ill-advised surgery or by autopsy, or by both. Pathologically the tube is but little thickened and may be entirely free from adhesions. Rarely is free pus found. Usually the induration is moderate and the fimbriæ are seldom involved. Involvement of the fimbriæ in some of these streptococcal cases may be the result of a preexisting gonococcal infection.

At this stage mention must be made of tubal infections secondary to neighbouring inflammatory conditions. The inflammation may result in peritubal adhesions which at a later date may give rise to the erroneous conclusion that the salpingitis is of primary origin. In the early stages, however, the tubes may be much enlarged, showing all the changes of an acute infection. The fimbriæ are not retracted and usually the primary disease is in evidence. In a low-lying appendix the tube may show greater reaction and the congestion of the appendix may appear to be secondary.

Chronic pelvic inflammation follows the acute and consequently has the same causative factors.

Curtis further considered the question as to whether the gonococcus produces a chronic salpingitis or whether it arises from repeated gonorrhœa of the tubes ascribable either to recurrence of infection from without or repeated invasion of bacteria from the chronically infected lower tract. He rarely obtained viable gonococci from patients who had been free from fever and leucocytosis for a period of more than two weeks, though good growths were obtained in recent tubal infections. He thus satisfied himself that the gonococcus lives but a short time in the tubes and that chronic salpingitis is not to be regarded as a form of chronic gonorrhœal infection, but the scene of invasion of bacteria from without or lower in the genital tract.

Collections of fluid in the pelvis which have by effluxion of time become sterile may become infected by the colon bacilli or other entero-bacteria and so cause pelvic abscesses of varying degrees of virulence. This will be more fully considered in the discussion on the treatment of chronic infections.

I have discussed the ætiology of pelvic inflammation at some length, for these organs, being easy

of access, serve, in the words of Hertzler, as "the happy hunting ground of the occasional operator". In the case of those falling into the gonococcal class, no great harm may arise from injudicious operative interference; but in the streptococcal infections of post-abortal or puerperal origin very serious disaster may occur from disturbing or setting free the more virulent streptococci into the peritoneal cavity.

Treatment.

Treatment must be considered under two heads, that of the acute and that of the chronic form of inflammation.

The Acute Form.

Dealing then with an acute inflammatory mass in the pelvis in which appendicitis and tubal pregnancy and suppurating tumour can be excluded, we have two modes of procedure: (i) either immediate operation, as advocated by Dubose, of Alabama, and others, or (ii) adopt a line of expectant treatment, when it will be found that operative measures will be required eventually to deal with the sequelæ of the infection in only a small percentage of cases. In other words, surgery should be directed to the reconstruction of tissues damaged by disease and not to stamping out the disease itself, so that the extent of operative procedures can only be applicable to each individual case.

The expectant treatment of acute pelvic infections can be considered with the following reservations. In the presence of a collection of pus low in the pelvis, or of an inflammatory mass in the parametrial connective tissue with severe progressive symptoms, provided the mass can safely be reached from below, vaginal drainage is the procedure of choice. Expectant treatment, then, is best considered under two headings: (i) general measures, applicable to most varieties of pelvic infection, and (ii) special measures, applicable to special conditions only.

The general measures applicable to practically all cases of acute pelvic inflammation should include the following: rest in the Fowler position, the giving of fluids, laxatives, necessary sedatives in case of severe pain, and antipyretics in moderation. Hot vaginal douches or Leiter's tubes are used. Hot applications may be given to the lower part of the abdomen, especially dry heat and diathermy, with such medical, serum or vaccine therapy as may be indicated.

Special measures indicated in certain cases of acute pelvic inflammation are most conveniently presented by stating the particular conditions for which they are used. If the infection has followed labour or abortion, it is most desirable to have the uterus clean. This result can be obtained in several ways, the most satisfactory being daily intrauterine applications of glycerine, and, if this fails to remove infective material, very careful curettage.

Again, if the infection has taken place through an operation wound of the cervix, remove the sutures so as to give free drainage to the inflamed area.

And again, if a collection of pus can be felt low in the pelvis, open and drain by posterior colpotomy. A word of warning may be given in regard to this. A fairly large butterfly drainage tube should be inserted and should be left in place till the cavity is nearly obliterated; gauze is unsuitable for keeping the track open, though it is useful to check hæmorrhage should it occur. Secondly, it is necessary to avoid irrigation of the abscess cavity, as the stream of fluid may break through some weak place in the protecting wall and spread infection to the general peritoneal cavity.

If the inflammation takes the form of a rapidly spreading virulent peritonitis, as seen principally in pelvic inflammation following labour or miscarriage, and if the patient is in a desperate condition, one is faced with the serious problem, whether to drain or not to drain. Abdominal section, under general anæsthesia, undoubtedly causes sufficient shock and lowering of the resistance of a patient already *in extremis* to go far towards bringing about a fatal result. In recent years the best results have been obtained in the Women's Hospital by concentrating on a policy of limitation of the infective process and elimination of the infective material.

By avoiding abdominal section the wall of exudate is preserved. The immobilization of the adjacent intestinal coils is favoured by leaving the adhesions and by quietening intestinal peristalsis through withholding food and, if necessary, by stomach washings and administration of repeated doses of morphine.

Elimination is advanced by a posterior colpotomy or insertion of a tube in one or both flanks under a local anæsthetic and thus securing exit for infected material aided by the Fowler position and free use of saline solution given by the rectum, and blood transfusion in cases of great severity.

The Chronic Form.

Turning now to the more chronic types of pelvic infections, those most in evidence are: (i) salpingitis (of varying degrees of intensity) with or without complicating oophoritis and pelvic peritonitis with exudate and adhesions, and (ii) chronic pelvic cellulitis consisting either of a walled-off collection of pus or simply a marked cellular infiltration causing induration and pain.

Chronic salpingitis follows the acute attack. If we are reconciled to the statement that chronic pyosalpinx, tubo-ovarian abscess, and hydrosalpinx are due to the gonococcus, then, knowing that the virulence of this organism in the course of a few weeks becomes so attenuated as to be practically negligible, we have good reason to treat all such cases expectantly in the acute stage, and, if no untoward symptoms occur in the chronic stage, to perform abdominal section at a considerably later period without incurring any risk to the patient.

Contrast this with adopting similar procedures in the case of chronic pelvic cellulitis. This also follows the acute condition and is usually due to infection following labour, miscarriage, operations on the cervix or within the uterus, instrumentation or attempts at abortion. If cellulitis alone (without tubal involvement) be usually due to streptococcus, practically never to the gonococcus, then the fact that streptococci differ from the gonococci in that they retain their virulence indefinitely indicates that expectant treatment should be carried out for a longer period, and abdominal section, even at a late stage, performed with considerable apprehension.

Fortunately, an abscess in the broad ligament or an inflammatory mass in the pelvic cellular tissue is either sufficiently low in the pelvis to be evacuated by a posterior colpotomy or extraperitoneally by an incision parallel with Poupart's ligament, as demonstrated with such clarity by Cullen,[2] of Baltimore.

With these brief notes as a basis for discussion, it is interesting to note the methods of procedure adopted by our colleagues overseas.

The Method of Priestly and Payne.

Priestly and Payne, in a paper read at a meeting of the Philadelphia Obstetrical Society in May, 1929, stated that:

> The conservative treatment of pelvic inflammatory disease is now the generally accepted method—operative interference in the acute stage is practised by few and the treatment of the majority of cases falls outside the domain of surgery; if necessary, the judicious conservation of the pelvic organs should always be the rule. For years the danger of operative procedures in the presence of acute infection has been recognized.
>
> Nevertheless, tubo-ovarian and pelvic abscesses will always require extra peritoneal evacuation and drainage.
>
> In chronic cases indications for operation are: repeated attacks, persistent adnexal masses with pain and tenderness, marked menstrual disturbances, etc. In the surgical treatment of such cases conservation of one or both ovaries and, if possible, the uterus, is the procedure for choice.

The Method of Hertzler.

Hertzler does not mix matters in regard to his views on the advisability of treating acute salpingitis expectantly or otherwise. His statement is that:

> Formerly acute gonorrhœal tubes were common objects in the laboratory, but since operations in the acute stage are no longer performed, they have become rare.

The Method of Aldridge.

Aldridge,[3] in a paper read before a combined meeting of the New York, Philadelphia and Boston Obstetrical Societies, states in his summary that abdominal operations for salpingitis, while the infection is still active, are accompanied by an unjustifiable mortality, excessive morbidity, especially from shock, sepsis and defective wound healing, a high percentage of radical surgery, and disappointing end-results.

Contrast his results in the following table, noting the very high mortality figures in the active type in comparison with the low percentage in the inactive class. The following table shows Aldridge's series of mortalities in 1,066 laparotomies for acute or chronic salpingitis, alone or associated with other pelvic pathological change.

TABLE I.

Type of Case.	Number of Cases.	Mortality.
Microscopically and clinically active	99	13·1%
Microscopically active, but clinically inactive	89	3·3%
Chronic inactive	878	2·8%

The Method of Green-Armytage.

Colonel Green-Armytage[4] has no hesitation in asserting that chronic pelvic suppuration is 80% gonococcal and 20% streptococcal, the former comprising tube and ovarian involvement, the latter cellular tissue. Out of 512 patients suffering from tubo-ovarian disease admitted to hospital during a course of five years, 373 were operated on, 269 by abdominal section and 104 vaginally. In the 269 laparotomies it was necessary to remove both tubes and partially resect both ovaries in 206, with a mortality of 6·3%. In 63 total or subtotal hysterectomies with removal of both tubes and ovaries there was a mortality of 9·5%. In 104 vaginal operations there was a mortality of 3%.

The Method of Dubose.

Dubose,[5] of Alabama, strongly disapproves of any delay in the surgical treatment of pelvic inflammation. He advises immediate operation in all cases which present the syndrome of acute surgical pelvic inflammation if first seen within twelve hours of the onset of the infection. Even if the *cul de sac* bulges with pus he does not hesitate to perform abdominal section, asserting that the greatest error in operative treatment is insufficient surgery and inadequate drainage. His figures illustrating 255 consecutive cases include 80 hysterectomies and 76 double salpingectomies with only one death. But as 156 women were sterilized out of 255 his method is too drastic for recommendation.

Results at the Women's Hospital, Melbourne.

Tabulating the last recorded 500 cases of pelvic inflammation treated in the Women's Hospital, we find the figures set out in Table II.

TABLE II.

Type of Case.	Number of Cases.	Treatment.	Total Mortality.
Pelvic cellulitis	58	Expectant	1
Pelvic cellulitis	22	Surgical	1
Acute salpingitis	188	Expectant	0
Acute salpingitis	10	Surgical	2
Chronic salpingitis	30	Expectant	0
Chronic salpingitis, including pyosalpinx, tubo-ovarian abscess *et cetera*	142	Surgical	6

The expectant treatment carried out was on the lines already detailed. The surgical treatment in

the case of pelvic cellulitis consisted either of vaginal or extraperitoneal drainage. In these cases of acute salpingitis with varying degrees of pelvic or general peritonitis treated surgically the procedure was vaginal drainage in four instances and abdominal section in the remaining six. In the 142 cases of chronic salpingitis alone or associated with pathological changes in other pelvic organs a posterior colpotomy was performed in 15 instances, salpingectomy with hysterectomy in 19, and in the remaining 108 unilateral or bilateral salpingectomy, including appendicectomy and oophorectomy when indicated. The mortality of 10 in a total of 500 cases results in a percentage very nearly approximating that obtained by Aldridge in his series.

Conclusions.

1. Assuming that in more than half the cases of chronic suppuration in the pelvis pus is sterile at the operation, sterilization of the infected focus takes place automatically within a reasonable time in the majority of cases.

2. Abdominal removal of the mass while the bacteria are active and virulent, as in the streptococcal group, results in fatal peritonitis or localized infection in many cases. Abdominal removal of the mass after the bacteria are dead or greatly attenuated is almost never followed by infection, even though there is an extensive escape of pus into the pelvis. Hence abdominal operation for a chronic inflammatory mass in the pelvis should not be undertaken during the period of probable sterilization, except in those rare cases in which, in spite of palliative measures, the patient's life is threatened by the severity of the inflammation and the infected focus cannot be drained extraperitoneally.

3. No intraperitoneal operation should be undertaken until the streptococcus is excluded with reasonable certainty. In a doubtful case in which the abdomen is opened on the supposition that the mass is tubo-ovarian and it is found before the adhesions are much disturbed that it is the connective tissue (parametric) which is involved, the route of attack should be changed to an extraperitoneal one (*per vaginam* or above Poupart's ligament) and the abdomen closed. Such a lesion probably contains streptococci and the adhesions of omentum and bowel which caused the deceptive mass high in the tubal region constitute Nature's barrier between the virulent bacteria and the peritoneal cavity; when the barrier is broken down the way is opened for a fatal peritonitis.

From this accumulation of evidence one feels justified in asserting that pelvic inflammation can be divided into two classes: (i) A gonococcal infection with resultant salpingitis. (ii) A streptococcal infection following labour or miscarriage or instrumentation or cervical erosions resulting in a parametritis or pelvic cellulitis with varying degrees of perisalpingitis. (iii) That acute pelvic inflammation gives best results when treated expectantly. (iv) That the surgical treatment of chronic infection should be undertaken for the reconstruction of diseased tissue and not for stamping out the disease itself.

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SOME NOTES ON THE EAR IN RELATION TO HEAD INJURY.[1]

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IN these days of motor car accidents and compensation for injury received at work, it is only natural that more attention is paid by the patient to any disturbance of hearing after a head injury than formerly; and with more prosperous times there must obviously be an ever-increasing number of these cases.

I offer this paper more to promote a discussion on certain aspects of these cases than to put anything more before you than a simple analysis of my cases together with some notes from the recent literature. Nearly all the patients in the series were seeking compensation for injuries causing deafness or dizziness.

In a series of forty consecutive patients suffering from head injury, with or without fracture, seen by me in my private practice and due to motor car smashes, falls on the head or severe hits on the head, and whose complaints of ear symptoms or dizziness were undoubtedly due to the accident, I find that in a large proportion there is deafness with a positive response to the Rinné test, that in the Weber test sound is referred to the good ear and that there is loss of bone conduction; in other words, that what we commonly refer to as nerve deafness is present. In these cases the deafness may be due to disturbances in the inner ear or to injury to the nerve by tearing, pressure from œdema or blood or, later, from overgrowth of scar tissue or possibly callus, or to some central lesion.

Unfortunately these patients are not as a rule referred to one till some time after the accident, that is, when they begin to think of applying for compensation; and very often the ear has not been

[1] Read at a meeting of the Section of Oto-Rhino-Laryngology, New South Wales Branch, British Medical Association, on March 20, 1934.

properly examined or tested until that time, the attendant at the time of the accident being content to look for bleeding from the ear and perhaps to make a brief note of "deafness present".

It would be of great advantage if these patients were seen by an otologist soon after the head injury occurred, particularly in those cases in which we later find a suppurating ear. It is often difficult to say later on whether the suppuration should be connected with the accident or not; whereas, if the ears were examined soon after the accident in all head injury cases a better understanding of the condition found later would be obtained.

Collie[1] points out that it is possible to tell whether a perforation is recent only if the ear is examined shortly after the accident. A recent rupture is fairly easily recognizable as such, but a rupture that is a week old is frequently indistinguishable from an old perforation due to disease.

However, when the patient is seen at a later date, certain points may help us in our determination of the amount of damage caused by the accident. For example, the state of the Eustachian tube in suppurating cases may help us in our decision. If it is examined fairly soon after the injury and is found to be wide, it is more probable that the suppuration has been caused by the injury. If the tube is narrow, it would tend to point more to some old-standing trouble, though, of course, the narrowness might be due to injury to the tube by a fracture passing along its roof.

The condition of the other ear is often of great help, as we may find an obvious old trouble in it, although the patient regards it as normal. If it shows a type of lesion which we might justifiably expect to find in both ears if one was affected, and if the injured ear has a similar appearance, we may more easily be able to estimate the amount of damage done by the accident to the ear about which the patient complains.

In my series I have not taken into account any cases of injury by penetrating wounds, burns, explosions or blows on the ear of a milder type which have apparently caused only rupture of the drum. I have considered only cases of definite head injury, and of these only those in which there was no doubt that the deafness had been caused by or increased by the injury, not necessarily a fracture.

Pathology.

The majority of fractures of the base of the skull involve the middle fossa. This is only to be expected, since a line of weakness extends through both petro-tympanic fissures and the Eustachian tubes to the *foramina lacera media*, between which are situated the sphenoidal sinuses. Most of the fractures of the middle fossa follow this weak line either wholly or in part. The thin roof of the canal of the *tensor tympani* and the Eustachian tube and that of the tympanum and mastoid antrum lie in this line and are almost invariably involved in this fracture. The extent of the injury will vary considerably with the violence and the site of application of the blow received.

The external auditory meatus is often involved in the fracture when the line is through the petro-tympanic fissure, and the fracture may split the meatus longitudinally, dividing it into an upper and lower half; or more commonly, a crack in the roof of the meatus is caused. In a few cases, when great force has occurred, the fracture may extend across the upper or anterior surface of the petrous bone into the internal auditory meatus.

Fractures of the posterior fossa commence in the thin bone of the cerebellar fossa and radiate into the lateral sinus groove of the same side, then pass along the groove of the inferior petrosal sinus or across the petrous pyramid near the inner edge of the lateral sinus, through the cancellous bone surrounding the mastoid antrum and the semi-circular canals, and behind and external to the internal meatus.

A moderate blow on the temporal fossa may produce a radiating fracture which cracks the roof of the middle ear, causing blood to effuse into the middle ear without rupture of the drum. This type of lesion would not be detected without an aural examination. No blood escapes into the external auditory meatus, but a typical bluish drum is said to be seen on examination. The only indication is deafness. In such cases blood might be seen at the orifice of the Eustachian tube. Davis[2][3] says that in this type of case hearing usually completely recovers in about three weeks. One or two in my series were evidently of this type and the hearing had practically returned to normal before I saw them.

Watkyn-Thomas[4] points out that in some of the cases in which blood is reported in the tympanum, the first signs are of middle ear deafness, but at the same time there is some blood in the perilymphatic spaces, and as this blood becomes organized into fibrous tissue, some nerve deafness may follow.

More commonly the upper half of the drum is ruptured and blood freely escapes from the external auditory meatus as well as through the Eustachian tube into the nose and pharynx, whence it may be expectorated. If the bleeding is accompanied by the leaking of cerebro-spinal fluid, the fracture is a severe and dangerous one, the dura having been torn or the labyrinth fractured.

Shemeley[5] states that about 80% of fractures of the base of the skull originate as a linear fracture in the vault and extend to the base of the skull. If this is so, we must expect the ear to be involved in a great number of these fractures.

Fractures of the pyramid may be classified into three groups:

(*a*) Longitudinal fractures. Longitudinal fractures start in the region of the *sella turcica* and pass backwards along the line of the spheno-petrosal fissure, breaking the roof of the Eustachian tube and tympanic cavity. The fracture may then

pass outwards to the external auditory meatus and squamous region. If this is the case the inner ear is not involved in the fracture itself, but the ossicles may be dislocated and the drum torn. However, damage may be done to the labyrinth by concussion. On the other hand, the fracture, after reaching the roof of the tympanic cavity, may pass inwards through the petrous pyramid and thus resemble those fractures which run at right angles to the long axis of the petrous bone. The inner ear is, of course, involved in these latter cases.

(*b*) Transverse fractures. Transverse fractures are less frequent. They always injure the labyrinth. As a rule the fracture passes through the external auditory meatus, roof of the tympanic cavity, vestibule and internal auditory meatus, as this is the line of least resistance. The fracture may, however, pass further forward through the cochlea or further back, when the canals are involved.

(*c*) Avulsion of the petrous tip. Avulsion of the petrous tip is very rare.

X ray examination is a definite help in establishing the fact that a fracture is present, but of less help in determining its extent. It is not wise to say that there is no fracture because it cannot be demonstrated in a radiograph.

Klingenberg[6] reports two cases of fracture of the petrous bone involving the cochlea and sparing the rest of the labyrinth, confirmed by X ray examination. There is complete deafness with preservation of the vestibular reactions. If, after injury to the skull, there is unilateral deafness with complete or partial preservation of the caloric vestibular reflexes, we can, in the absence of bleeding from the ear or injury to the middle ear, and supported by a positive X ray finding, diagnose with certainty an isolated fracture of the cochlea. The functional disturbance alone might arise from a tear of the cochlear nerve, which would be attended with less danger to life.

In those fractures which involve the petrous pyramid, the labyrinth capsule may be rent and the membranous labyrinth torn, but more frequently the damage is limited to hæmorrhage into the labyrinth spaces without fracture of their bony walls. This naturally injures their end organs, often very seriously. In addition, they may be disorganized subsequent to injury by the formation of callous or scar tissue. On the other hand, effused blood is sometimes absorbed in the process of healing, and thus the patient may regain some of the hearing power.

In addition to, or independent of, damage to the labyrinth proper, the auditory nerve trunk may be ruptured by the fracture or compressed by the extradural hæmorrhage or by œdema, and the same is true of the facial nerve.

Lannois, Chavanne and Bourgeois[7] state that though fractures, great and microscopic, naturally do occur, they think that rupture of minute blood vessels or tearing of nerve filaments, either of which might cause destructive or degenerative changes within the labyrinth, may be caused by cranial injuries which produce absolutely no loss of continuity of the labyrinth capsule.

Stenger also notes that the membranous labyrinth may be damaged by violent disturbances caused by trauma to the head, even though the temporal bone is not fractured.

Grove[8] declares that in the temporal bone most autopsies show that hæmorrhage is the salient feature. Unless the labyrinth capsule is fractured, such hæmorrhage is always perilymphatic, the usual site being in the *scala tympani* near the round window.

The nerves may be damaged by pressure from hæmorrhage: (i) before entering the pyramid, (ii) within the porus, (iii) in the terminal canals.

Grove found in patients dying years after the injury, atrophy of nerve fibres and of the organ of Corti, especially in the basal coil, and more or less complete filling of the inner ear spaces and canals with hyaline tissue and bone. In this connexion he points out that middle ear deafness, such as may follow a longitudinal fracture, can improve; but, on the other hand, when perilymph or endolymph has been disturbed or replaced by hæmorrhage, a progressive type of deafness follows.

Cases of labyrinth injury associated with fracture of the base may be divided into recent and old ones. In the former we have marked hæmorrhage into the inner ear. Even when the labyrinth capsule is not injured, we meet with small hæmorrhages in the inner ear and eighth nerve, which we may possibly find ruptured. In old cases the line of fracture may be visible, partly closed by new bone and partly by fibrous tissue. In others again the line of fracture is not evident.

Barnick reports four cases of fracture of the base of the skull in which the labyrinth capsule was not affected. In these cases hæmorrhages were found in the perilymphatic spaces, in the acoustic nerve and in its branches.

Several cases have been reported of fracture of the base of the skull in which the auditory nerve in the internal auditory meatus was torn without any injury to the labyrinth capsule.

Damage to the brain tissue may be caused by direct injury or *contrecoup*. Grove believes that compression of the lateral ventricles may drive the cerebro-spinal fluid into the fourth ventricle with sufficient force to damage the vaso-constrictor centres and so produce stasis in the brain tissue with injury to the central vestibular area.

In animal experiments he has shown changes in the nuclear region of the eighth nerve in the floor of the fourth ventricle, mainly affecting the cells of the superior vestibular nucleus, the *area acoustica* and the *corpora quadrigemina*.

Newton Evans and Courville,[9] in studies on the histological changes in the brain following head injury, have found small petechial hæmorrhages in the central nuclei of the acoustic and vestibular nerves. In the nerve cells comprising these ganglia

they have observed vacuoles in the nuclei and cytoplasm, and suggest that these vacuoles indicate damage to the ganglia along with the rest of the brain, that is, generalized œdema, and that more or less permanent injury may result from these changes.

Recently Wittmack,[10] both by animal experiment and by *post mortem* examination, has shown that head injuries, particularly those due to blows in the occipital region from falls, may cause a complete nerve deafness on one or both sides without concussion or fracture of the base, due to destruction of the organ of Corti in all the coils. The sacculus is also destroyed, but the utriculus and other vestibular end organs are unchanged. The cochlear ganglion in the spiral lamina shows a marked diminution in the number of cells, and the cochlear nerve is also atrophied later.

To state his explanation briefly, he says that when a compression shock on the endolymph, transmitted through the bones of the skull, attains a high degree, the protoplasmic contents of the organ of Corti are crushed between two independent systems of pressure of the endolymph, and the whole end organ may be very suddenly and completely destroyed.

Thus the common causes of destruction or injury to the labyrinth resulting from accident[11] are: (i) fracture involving the labyrinth, (ii) hæmorrhage into the labyrinth, (iii) concussion sequelæ.

Fracture and hæmorrhage in a large number of cases totally and permanently destroy both cochlear and vestibular functions, while concussion may not be of such severity as entirely to destroy both these functions, but may permit some portions to escape. As Fisher points out, concussion may produce all sorts of freakish phenomena.

A severe injury to the vestibular apparatus practically always involves the cochlear apparatus and usually results in total, or very extreme, deafness on the side affected. On the other hand, a severe injury to the cochlear apparatus does not necessarily cause a severe injury to the vestibular apparatus.

This is helpful in dealing with some cases. The vestibular responses are objective and cannot be affected in any manner by the patient, and a definite lack of all response to stimulation indicates a definite loss of function. Finding this, it is fair to assume that the cochlea, the less resistant organ, has almost certainly suffered the same fate. Thus, if we find total loss of vestibular reactions on one side and the patient is apparently totally deaf in the corresponding ear when we test it, there is little need to consider the question of malingering. On the other hand, when we have the vestibular system reacting normally but total deafness is complained of on that side, we must always test carefully for malingering. In the cases of malingering that I have come across, I have always found that the patient complained of total deafness in one ear.

Signs and Symptoms.

As stated above, fractures of the base of the skull more often involve the middle fossa. This injury, if the petrous portion of the temporal bone is involved, often produces several signs of diagnostic importance:

1. Escape of blood or cerebro-spinal fluid, or both, from the external auditory meatus or from the Eustachian tube.
2. Deafness, more or less complete and of sudden onset.
3. Facial paralysis.
4. Vertigo.
5. Spontaneous nystagmus with the quick component away from the side injured, if the patient is conscious. If the patient is not conscious, then there will be divergence of the eyes towards the injured side,[12] if the labyrinthine lesion is an irritative one.

Injury to other cranial nerves may occur, but much more rarely.

In the series of cases under review a history of the first four of these signs or symptoms is of importance. The fifth does not concern us so much, as it is one which has usually disappeared before the patient is seen by the otologist, and information on it cannot be obtained from the patient and sometimes not from the records.

Tearing of the tympanic membrane may result from fracture and produce bleeding from the ear. Occasionally, however, the bleeding from the ear may be due to an injury to the external auditory meatus alone. This blood may be mixed with cerebro-spinal fluid. We must here remember that many who see a patient after an accident with profuse sanious discharge from the ear, are apt to conclude that it is cerebro-spinal fluid without a proper investigation.

Hæmorrhage from both ears is always an unfavourable sign, and the mortality is stated to be about 66%, as against 39% in those with hæmorrhage from one ear only.

The deafness in these cases of head injury, I have found, is usually of the nerve variety. It is frequently severe and sometimes absolute. Any improvement in hearing, according to several observers, usually becomes manifest in about eight weeks after the injury, and if deafness remains after that period it is likely to be permanent, although Delie[13] had a case with recovery after as long as one year.

The deafness is often accompanied by tinnitus, which sometimes persists for twelve months or more. The lesion is not always in the internal ear. It might be of central origin, due to injury of the brain or central nervous system.

Concussions of the head have a deleterious effect in those cases in which an aural affection, associated with disturbances of hearing, has already existed, as even very slight shocks suffice to bring about an aggravation of the latter. Most of my cases in

which there was any evidence of old trouble showed a marked to severe disturbance of hearing.

The facial nerve may be involved in the fracture as it passes through the facial canal, particularly near the geniculate ganglion. The onset of the paralysis is almost immediate, but if it is delayed it is probably due to hæmorrhage into the facial canal or to pressure from œdema, following which recovery of the paralysis is more rapid. All patients seen by Davis[14] with facial paralysis recovered, even after long intervals, although he states that in two cases a facial tic or twitch persisted.

Vertigo[15] is sometimes experienced for a long time after the accident. The disturbances of hearing are more conspicuous than the disturbances of equilibrium in lesions of the inner ear resulting from direct cranial injury. This does not mean that the static labyrinths do not suffer, but that by comparison with the deafness the disturbances of equilibrium often seem inconspicuous. Naturally these cases as a rule exhibit disturbances of equilibrium in the days immediately following the injury, but even when there is permanent and absolute deafness, Bourgeois believes that the vertigo, subjective and objective, disappears wholly in time. Dundas-Grant says that the tinnitus and vertigo are often as much due to concussion as to local damage to the ear. Following a fracture of the skull, involving the temporal bone, there can be vertigo with normal Bárány reactions.[16] Grove states that caloric tests showed a normal response in ten of his twenty-eight cases in which vestibular symptoms were present, that is, in about 36%. In my series I found an even higher percentage than this, namely, 50%.

The vestibular symptoms developing after injury to the labyrinth have been distinguished by Bárány as: (i) cases with complete destruction of the cochlear and vestibular apparatus following a fracture of the pyramid, (ii) cases in which the irritability of the vestibular apparatus is preserved and there are attacks of dizziness. In such cases the hearing may be more or less impaired, but cases are met with in which the hearing power is normal, or nearly normal. The attacks of dizziness appear especially when the head is rotated rapidly, when bending over, and during physical exertion. They may come on without any external cause, and the symptoms often last for years and incapacitate the patients for long periods.

In a few cases slight injury may be followed by intense vertigo when the head is in certain positions only. Watkyn-Thomas suggests that in these cases there is probably some injury to the otolith membrane.

Taking it all round, the fracture itself, which occurs frequently in severe head injuries, is only of potential importance; the essential point is the injury to the intracranial contents, and as a matter of fact the fracture may even limit the amount of damage done by the relief of pressure. However, from the otological point of view, of course, if the fracture involves the petrous bone, it may be of more importance than any general concussion lesion.

Patients who remain unconscious for hours or days are suffering from contusion of the whole brain, with petechial hæmorrhages and generalized œdema.

No concussed person, as a rule, ever remembers the exact details of his accident. He will remember things up to a point, but not what happened at the moment he was struck, and Trotter says that no lapse of time will ever bring that missing period back. The occurrence of this amnesia when examining the patient later is of importance, for by it we may judge whether the patient was concussed or not. Also the period of unconsciousness following the accident may help us in judging the severity of the injury he received. We must remember, however, that cases have been reported in which an injured person went about his work with a fractured base and no loss of consciousness.[17]

This question of amnesia is also of importance when we suspect malingering, for if the patient states that he was unconscious for some long time and then describes minutely the details of the actual accident as distinct from the events leading up to it, he should always be looked on with some suspicion.

In addition, though there is no direct relation between the severity of the injury and the occurrence or degree of vestibular or cochlear symptoms, nevertheless the history of the general injuries sustained in the accident is often useful.

Injuries to the nerves, such as those evidenced by total deafness, facial paralysis, paralysis of the abducent nerve *et cetera*, are a help in the diagnosis of fracture, but it must be borne in mind that any of these can be caused by injury to the substance of the brain without fracture.

Complications.

Suppuration may develop following a fracture involving the ear. This may result from: (i) infection through the Eustachian tube, or (ii) contamination of the extravasated blood through the external auditory meatus.

Should this infection occur, an acute mastoiditis may develop and this may require very urgent operation.

Another danger to the patient if suppuration of the middle ear ensues in a case of fracture involving the temporal bone, is the possibility of meningitis developing. There is increased risk of this in cases in which suppuration existed before the injury.

If the patient recovers from the immediate effects of the accident, he is not out of danger, as the onset of meningitis may be delayed for quite a considerable time, and as the fracture heals by fibrous rather than by bony union, later infection may travel up this path. Fraser[18] has reported a case of meningitis occurring twelve months after the original fracture.

Two cases of interest are reported by D'Onoforio.[19] In the first, the patient was hit

on the mastoid. All signs of the injury cleared up, but twenty days later he developed an acute mastoiditis with involvement of the middle ear. The effused blood had evidently become infected.

The other patient received a blow on the mastoid from a packing case. The next day the mastoid region was swollen and painful, and these symptoms increased for a fortnight, by which time they had become very severe. The middle ear was quite normal. Exploration revealed a small fistula through the tip leading into a cell filled with pus and granulation tissue with inflamed bone around it. The antrum was not affected. The damaged tissues were apparently infected from the blood stream.

One of my cases was somewhat similar to this, D'Onoforio's second case.

The patient had a severe blow on the mastoid and developed severe pain and congestion of the mastoid region. He had slight dulness of both drums, evidently from some old-standing trouble, with some deafness, more marked in the injured ear, which also showed some loss of bone conduction. X ray examination revealed a slight haziness of the mastoid cells on the injured side. There was no rise of temperature, and at no time were there any signs of congestion about the middle ear. After some days the condition settled down, the hearing remaining the same.

In this case the damaged tissues did not become infected after the injury.

Other complications which may develop are reported, but are very rare. For example, a case of temporo-sphenoidal lobe abscess is reported by Grehe, and a case of traumatic meningocele of the external auditory meatus following fracture of the skull is reported by Rollin.[20] Jenkins describes a case in which an abscess formed in the squamous portion of the temporal bone after a fractured base.

Analysis of Cases.

The following is an analysis of my cases. They are made up of ten motor car or cycle smashes, sixteen falls on the head from varying heights, and fourteen hits on the head, making a total of forty cases.

I find that the time at which these patients were seen after the accident varies from two weeks to five years and five months, a fair average time being about six months after injury. There were thirty-seven male and three female patients, and their ages varied from twenty-two to sixty-three years, the average being thirty-six years.

Thirty-one patients, or 78%, were rendered unconscious by the accident. The time of unconsciousness varied from a few minutes to three weeks. An analysis shows that, while one cannot definitely say that the longer the time of unconsciousness, and presumably the greater the damage to the head contents, the greater will be the damage to the hearing, still on the whole it appears to follow that it is so. For example, of those who had a prolonged period of unconsciousness, 76% had a marked degree of deafness and only 24% a moderate or slight degree.

Thirty-eight patients, or 95%, complained of deafness; the other two complained of dizziness, but deafness was also found to be present on examination, but only to a slight degree. In the one in which it was most marked the labyrinths were normal on testing. In the other there was only a very slight diminution of hearing in one ear, but there was a definite disturbance of the labyrinths on testing. However, when this patient was reexamined two months later, his labyrinths gave equal reactions.

Four patients, or 10%, complained of deafness in both ears. Of the remainder it is of interest to note that 35% had deafness in the left ear, while 65% had deafness in the right ear, that is, a proportion of nearly two to one in favour of the right ear being affected.

In most of the cases the deafness was noticed soon after the accident, when the patient regained consciousness.

One man, however, who had had dizziness, getting worse, ever since his injury (he had been hit on the head by a motor car), developed deafness in both ears three months after the accident. His serum gave a positive Wassermann reaction, and his lesion, while no doubt a specific one, was, one felt, related to the disturbance caused by his head injury.

In only two cases was any improvement in hearing admitted to. In both of these there had been a rupture of the drum.

The patients were seen three and five weeks after the accident respectively. The hearing had almost completely returned to normal in the first, though there was a severe injury involving the facial nerve and abducent nerve paralysis was also present. There was a loss of only about 5% of hearing in the second case. In the case with abducent paralysis X ray examination revealed a fracture extending from the left parietal region to the tip of the petrous bone. These patients had both apparently had a middle ear type of deafness with no loss of bone conduction. There had been discharge from the ear in the second case for the first three weeks following the injury.

On making a classification of the types of deafness, I find that 35% had a middle ear type of deafness, while 65% had a nerve type, that is, they gave a positive response to a Rinné test, had loss of bone conduction *et cetera*, or else total deafness with the tuning fork on the mastoid referred to the good ear. Hence there was a marked preponderance of nerve type deafness among these patients. In fifteen cases I was unable to demonstrate any hearing in the ear affected. In these the tuning fork was usually referred to the other ear when placed on the mastoid, and they were all carefully tested for malingering.

Tinnitus was complained of in 62·5% of the cases. Neither the degree of deafness nor the period of unconsciousness appeared to have any bearing on the presence of tinnitus. In only two cases did the patient admit to any improvement, but of this group with tinnitus 76% were seen within four months of the accident, which was perhaps too soon to allow any opinion to be formed as to the prognosis in regard to the tinnitus. Both the patients who admitted to improvement had a middle-ear

type of lesion and were seen within five weeks of the accident.

Tinnitus was complained of in almost the same proportion in both the nerve and middle ear types of deafness, that is, in 68% of the former and in 57% of the latter.

Fourteen patients, or 35%, gave a history of bleeding from the ear.

One patient had a history of bleeding from both ears, and he, incidentally, had had no loss of consciousness, but there was complete deafness in one ear while the other was normal on testing. From the deaf ear he had had some slight discharge for a few days following the injury.

Of these fourteen patients, nine had middle ear type of deafness, one had partial nerve deafness, and four had total deafness in the ear affected. Thus, in those with bleeding from the ear, the largest proportion, namely 64%, had middle ear type of deafness. Curiously enough, I find also that of those with middle ear type of deafness 64% had bleeding from the ear.

Eleven patients complained of pain in the ear on occasion, but in most cases it was not very severe.

Only five patients, or 12·5%, had discharge from the ear. Four of these had had bleeding from the ear at the time of the accident and in the other case there was no history obtainable regarding any bleeding. In three of these cases the discharge ceased soon after the accident. In one it had dried up two months after the injury, but had returned and was present when I saw the patient two years later. In the other case there was a certain amount of doubt as to whether there had been discharge before the injury. The major trouble to the ear, however, had certainly been caused by the accident, as the patient had total deafness in the ear when examined. Three of these patients had a fracture demonstrable by X rays, but in one the fracture was on the side opposite to the discharging ear.

There were undoubted signs of previous aural trouble present in thirteen cases, or 32·5%.

There were six patients with facial paralysis; four of these had improved when seen.

One of these four had fallen from his cart, being unconscious for four days. He had had bleeding from the right ear, which, when seen by me two and a half months later, had practically no hearing in it. He had good hearing in the left ear, but since the accident had had left facial paralysis, which was improving when I saw him.

Of the other two patients, one was seen only two weeks after the accident and the other was seen six months afterwards, but I find I have no record as to the amount of improvement or otherwise among my notes in this case. From this it would appear that the facial paralysis in these cases generally tends to improve.

There was only one case in the series with abducent paralysis. This followed on fracture of the tip of the petrous bone, and the patient had normal hearing within two weeks of the accident after the removal of some blood clot from the meatus.

Headache was complained of in twenty-seven cases. Nine of these patients were seen more than six months after the injury, and one as long as three years afterwards. Only five patients admitted to any improvement in the headaches, and in these instances the headaches improved within the first few months.

In one case only was there any definite spontaneous labyrinthine nystagmus seen on examination, and only two patients had signs of spontaneous disturbances of equilibrium.

Thirty-two patients, or 80%, complained of having had dizziness, but six of these noticed only a slight degree on first getting out of bed, thus leaving twenty-six patients, or 65% of the total, with a definite complaint of dizziness following the accident. Of these, 69% had nerve type of deafness and 31% middle ear type. However, on working out the percentage for each type of deafness we find that dizziness occurred about equally in each type, that is, 69% of those with nerve deafness complained of dizziness, while 66% of those with middle ear deafness had this symptom.

This dizziness is usually complained of on stooping and getting up suddenly, and on unusual quick movements *et cetera*. It had improved in ten of the cases and disappeared entirely in one, which was in the middle ear group, after inflation. In only one case was it stated to be getting worse, and this was in the syphilitic patient referred to previously. Those cases which showed improvement were equally divided amongst the patients with nerve and middle ear type of deafness, for 38% of the former and 37% of the latter showed improvement.

The sensation described varied from a sensation of floating on a board on water, or a sensation of themselves turning to a definite rotation of objects. Most of the cases were of a mild degree, and the dizziness was described as a swimming in the head.

Only five patients stated that the giddiness was so bad at first that the head had to be kept still while they were in bed, and of these three had no reaction of the labyrinth on one side, while the other two had normal labyrinths.

Seventeen patients gave a history of vomiting. In seven of these it was definitely related to the dizziness, while the other ten did not connect it with any dizziness which they had had, two stating that they had had no dizziness.

Nineteen patients, or 47·5%, complained of unsteadiness, but it was mostly a tendency to fall forwards, only seven giving a history of falling to any particular side. There would appear to be no definite relation as to the direction of the fall and the ear injured. Of the seven patients, three fell towards the side of the ear affected and two away from it. Two were uncertain as to which side they had fallen, though they were certain it was to the side and not backwards or forwards.

Seven patients, or 14·5%, gave no reaction to the caloric test. All of these had more or less severe

dizziness at the time of the accident, except one. This one, while he had had no dizziness, had had very severe vomiting when first in hospital. Thus 27% of those complaining of dizziness had no reaction obtainable in the injured labyrinth.

Of these seven patients four had no hearing in the corresponding ear, and the other three had very marked deafness.

Of the fifteen patients with total deafness eleven complained of dizziness. Of these fifteen patients thirteen had the caloric test done. Four had no vestibular response to the test, while nine reacted quite normally. Of the other two patients, one had never had any dizziness at any time, and the other, though suffering from repeated dizzy attacks, refused to submit to the test.

Thus it will be seen, as stated above, that the cochlear nerve appears to be the less resistant. A severe vestibular nerve lesion is usually associated with a severe cochlear nerve lesion, but a severe cochlear nerve lesion is not necessarily associated with a severe vestibular nerve disturbance.

There was some difference in the caloric tests on the two sides in six cases, but two of these eventually gave a normal reaction in one and two months respectively. Three of the remaining patients had some old-standing ear trouble, which in one instance, I think, contributed to the difference between the two ears when the test was carried out. These patients also showed corresponding disturbances in the rotation tests.

In 50% of the patients complaining of dizziness there was on the first examination no abnormality in the results to the labyrinthine tests. In other words, of those complaining of dizziness 50% gave normal responses to the caloric and rotation tests.

Of those who admitted to improvement in their dizziness, three had no reaction to the caloric test, and in the other six the result of this test was normal, though one of these at first showed some abnormality between the two sides.

The improvement in the dizziness was fairly equally divided between those in whom the labyrinths were normal on testing and those in whom there was some abnormality, for of the latter 40% and of the former 44% showed improvement.

Two patients gave a history of syphilis. In these the injury apparently stirred up the trouble in one, while in the other the syphilitic condition did not appear to have any relation to the trouble found, which was total deafness and total non-irritability of the corresponding labyrinth, with no alteration under antispecific treatment.

X ray examination revealed a fracture in thirteen cases. No fracture was detected in four. In the others either no X ray examination was made or I was unable to obtain a report.

In considering these cases it must be recognized that it is not so much a series in which there is deafness after the head injury, but a series in which there is deafness or dizziness persisting for some time after the head injury. If all cases in which deafness after a head injury is complained of, were taken in a series doubtless there would be a bigger proportion of middle ear involvement, as well as a bigger proportion of those with improvement. However, from this present series, in which the patient was as a rule not seen for some time after the accident, it would appear that a large number of patients may have a nerve type of deafness after a head injury.

Conclusions from Analysis of Cases.

1. The greater proportion of patients with persistent deafness after head injury have a nerve type of deafness.
2. A large number of these patients complain of tinnitus.
3. Only a small percentage have suppuration of the middle ear following trauma from head injury.
4. Facial paralysis, when it occurs, tends to become less, and generally to disappear.
5. A large number of patients have dizziness, but of these a large proportion show no abnormality to the caloric and rotation tests.
6. The dizziness tends to become less, but may persist for a long time in some cases.

Points for Discussion.

Points that I should like particularly to bring up for discussion and on which I should like to hear the opinion of members are:

1. The type of deafness most commonly seen after head injury, that is, is it more frequently nerve or middle ear type.
2. The prognosis as regards: (*a*) hearing in both types of deafness, (*b*) tinnitus, (*c*) facial paralysis, (*d*) dizziness. In those cases in which there is total destruction of the labyrinth we should expect that the patient would eventually regain his labyrinthine balance. When there is partial destruction there will probably always be some dizziness. There are still, however, a large number of patients complaining of dizziness in whom we find normal caloric and rotation responses. What is the prognosis as regards dizziness in these cases?
3. What should be our attitude in regard to allowing the patient who has had dizziness to return to work? Should we permit a man who has total destruction of one labyrinth to return to work where a fall may endanger him after he has apparently regained labyrinthine balance? What should be our attitude in regard to cases where dizziness is complained of and the reactions, as elicited by the caloric and rotation tests, are normal? When can we say they are fit for work in the general labour market? Perhaps, though, these latter cases, after we have demonstrated that the labyrinths are normal, come more under the jurisdiction of the neurologist.

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Reviews.

TUMOURS OF THE BRAIN.

So great have been the advances in our knowledge of brain tumours and in neuro-surgical methods of diagnosis and attack, as a result of the unique and painstaking work of Harvey Cushing and his associates in the last two decades, that a comprehensive review of the present-day position with regard to these tumours has long been looked forward to by those interested in these problems. Who should be better fitted to propound this information than Professor Percival Bailey, one of his master's most distinguished pupils? He has already written extensively on isolated sections of this subject, and his latest book, "Intracranial Tumours",[1] is a simple and complete exposition of a complex subject.

Tumours developing within the cranial cavity are many and varied; somewhat less than 50% occur in the cerebrum, so that the term "cerebral tumour" should no longer be used as a generalization. Similarly, discussions of intracranial tumours as a whole are useless and reminiscent of the days when it was taught that the cardinal symptoms of tumour of the brain were headache, vomiting and defective eyesight. These are merely the symptoms of increased intracranial tension, and the existence of a tumour should be diagnosed in ever-increasing frequency before their appearance. When confronted with a case of steadily increasing alteration of nervous function, the earliest and most important symptom, a threefold diagnosis should be arrived at without delay, namely: (i) the location of the lesion, (ii) its nature, (iii) if a tumour, its pathological type. In most cases the clinical methods of inspection and palpation are not applicable, as they are in diseases of the abdomen, and resort must be had to observing disturbances of intracranial physiology to localize the lesion. This should be possible in about 85% of cases by clinical methods alone.

To be able to form a pre-operative opinion of the pathological nature of the tumour is of utmost importance to the neuro-surgeon, because each variety presents its own difficulties and indications for removal, and its own prognosis. And it is primarily from this pathological standpoint that Bailey has written the present publication, amplified from the lectures and demonstrations given to his students. As these form part of the general course in neurology, the syndromes caused by certain tumours are used to illustrate much of pure neurology and some apparently extraneous sections have had to be introduced.

The general plan of the book is a well arranged and comprehensive one. Tumour is used throughout in its more restricted sense of a neoplasm, and the opening chapter provides a summary of the present-day position with regard to the ætiology of tumours in general and brain tumours in particular, which now represent 1·8% of all newgrowths. The complete classification given of these tumours, according to their tissue of origin, into twenty-eight types is the generally accepted one, and statistics showing the frequency and age periods of the various types are included. The details of the subarachnoid pathways, ventricular system, and full descriptions of the cortical convolutions, with their arterial supply and venous drainage, are splendidly set out in the next chapter on anatomical considerations. Although modern operative methods of exposure no longer call for such complicated measurements of cerebro-cranial topography as are supplied in the anatomy books under Chiene's and Reid's methods, some such simple system as is provided by Taylor and Haughton's lines, might well have been included here.

The third chapter deals with the physiology and the localization of function in the brain in a most thorough manner, especially with the effects of arterial and venous occlusions and injuries to certain areas. These all go to show that the brain cannot be insulted with impunity. We are in entire agreement with the warnings here uttered and frequently repeated to the students, especially not to be misled by reports of removal of the frontal lobe or of other parts of the brain without resultant symptoms, because should such a mutilation be tried symptoms may be discouragingly persistent.

The succeeding chapters deal with the origin, structure, symptoms and treatment of each pathological entity. Actual clinical cases and records of each variety of tumour are cleverly introduced and chosen to illustrate the various syndromes associated with the classical sites and differences of localization. Thus, under the craniopharyngiomata, is discussed the newly recognized and important syndrome of the hypothalamus; the pinealomata explain the syndrome of decerebrate rigidity; the medulloblastomata illustrate the syndrome of the vermis of the cerebellum, whilst the astrocytomata describe the distinct, though associated, syndromes of the cerebellar hemispheres and the frontal lobes, and so on.

It is difficult to select any particular portions for special mention. The section on the intricate subjects of aphasia and apraxia, illustrated by actual cases, is a particularly good one, and so is the section on the histogenesis of the encephalic gliomata, as is to be expected in view of Bailey's previous work on this subject. His differential diagnoses of unilateral exophthalmos and intracranial calcification are also well arranged; and his ideas on the morphology and ontogeny of the cerebellar divisions are well conceived.

The chapter about tumours of the pituitary gland itself deals comprehensively with this complicated subject from embryological, histological and semeiological points of view; but it is rather surprising not to find any mention of the recently described syndrome of the basophile adenomata, nor of the production of prolan *A* and *B* by the anterior lobe, with its bearing on the Aschheim-Zondek test for pregnancy. Perhaps also the transsphenoidal operation might have been discussed as a decompressive measure.

[1] "Intracranial Tumours", by P. Bailey; 1933. London: Baillière, Tindall and Cox. Crown 4to., pp. 500, with illustrations. Price: 35s. net.

The concluding chapters are concerned with general and differential methods of diagnosis and with the broad details of pre-operative and post-operative treatment and operative technique. We fully endorse the stress the author lays on the value of a true chronological record of the occurrence of the symptoms, of ophthalmoscopy and of accurate and repeated perimetry. The uses and abuses of encephalography and ventriculography are fully pointed out. The pitfalls produced by primary vascular lesions and by syphilis of the brain receive ample consideration; warnings are given of the frequent fallacy of diagnosing migraine and epilepsy in persons of middle age without any hereditary taint; and attention is drawn to the increasing occurrence of pulmonary cancer, whose intracranial metastases are often manifest before the primary lesion is diagnosed. The very detailed classification of the types of encephalitis seems rather unnecessary. It is interesting to note the author's advocacy of the intratracheal administration of ether when general anæsthesia is necessary, and his admission that it is often not given because a good anæsthetist is not available. The great advantages and maximum safety of this type of anæsthesia have long been recognized in this country, although it is definitely contraindicated in cases with high intracranial tension, arteriosclerosis and syphilis. His advice against the use of preliminary medication, especially with morphine, "Amytal" and "Avertin", is strongly endorsed, as they are depressants of the nervous system generally and of the respiratory centre in particular; and we also agree with his cautionary remarks about the indiscriminate use of the electric current as a hæmostat. With regard to results of surgical treatment, we join with Professor Bailey in reminding the critics that the operations are for "cancer" of the brain, and that they are about as successful, finally, as for cancer elsewhere in the body; and that it is a precarious, back-breaking, heart-rending business, to be undertaken only by those who have especially prepared themselves for it.

The author is to be congratulated on the extensive bibliography, with references in the text, which contains only those publications written in English, those written in a foreign tongue, whilst of acknowledged merit, not being considered accessible to the average student whose time and linguistic ability are limited; also on the choice of dull paper in place of the usual art paper used recently in most medical books, which tends to blind the reader. The pen and ink drawings and the semi-schematic reproductions of skiagrams are quite satisfactory, the only really adequate way to demonstrate the latter being to bind the actual photographs into the book and supply a stereoscope for their examination.

TUMOURS OF THE SKIN.

MEDICAL books by Australian authors who can speak with authority on the subject discussed are gradually increasing in numbers. It is a pleasure to add in this category, Dr. C. Norman Paul's "Cutaneous Neoplasms".[1] It is nearly sixteen years since he published his work on the influence of sunlight in the production of cancer of the skin, and in the preface to the new volume he utters a timely warning to sun-bathers against reckless exposure to sunlight. This warning necessarily applies more to Australian conditions than to British. There can be no doubt that our climatic factors play an important part in the genesis of skin lesions, some of which may become malignant, and that Australian dermatologists have, in consequence, unrivalled experience in the study of these. Dr. Paul has embodied part of his own extensive practical knowledge of the subject in the work before us for the benefit of his fellow practitioners, Australian more particularly. The patient with a skin lesion usually consults first his own medical attendant. In the capital cities he is then, after a preliminary diagnosis, usually referred to the dermatologist. In the country the general practitioner may have to play a more active part. He will often find great difficulty in determining the exact nature of the lesions presented, and we think that this work with its sixty-two illustrations and moderate price will be of definite assistance to him. The following list of some of the subjects discussed and figured will indicate the scope of the work and show that it is by no means confined to true neoplasms: chronic solar dermatitis, arsenical keratoses, *xeroderma pigmentosum*, rodent ulcer and epithelioma and allied neoplasms, secondary carcinoma, Paget's disease of the nipple, cutaneous horns, melanomata of the skin, chronic nodular chondrodermatitis of the helix of the ear, seborrhœic warts, various tumours, cysts and anomalies of the sebaceous and sweat glands, Brooke's *epithelioma adenoides cysticum*, Bowen's disease, *granuloma pyogenicum* and *granuloma annulare*, neurofibroma, cheloid and colloid degeneration of the skin.

We miss any reference to *mycosis fungoides*. Dr. Paul considers, contrary to the views of some, that trauma may occasionally be the factor that is responsible for the development of a rodent ulcer, and gives as examples a scratch from a hatpin, bites from parrots and kookaburras, pressing out comedones and various injuries. He emphasizes that great care and caution should be exercised in making biopsies for diagnostic purposes, and would veto these in such situations as the naso-facial and naso-labial junctions where cartilage so closely underlies the skin. In conclusion, we would again call the attention of the general practitioner to the contents of this work as we think that it may be of real assistance to many.

[1] "Cutaneous Neoplasms", by N. Paul, M.B., Ch.M.; 1933. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 168, with 62 illustrations. Price: 10s. 6d. net.

Notes on Books, Current Journals and New Appliances.

A MEDICAL DICTIONARY.

THE twelfth edition of Stedman's "Practical Medical Dictionary" is to hand.[1] This book is well known and needs no introduction to medical readers. It is, of course, American, though published by a London firm. In this edition thirty-three pages have been added to the last, and all the changes in the British Pharmacopœia have been noted. In a search for omissions the only one of importance is the Casoni test; this surely is worthy of mention. The omission of the Bendien test for cancer is possibly justifiable. This book may be recommended as a reliable dictionary to students and practitioners.

CORPULENCE.

OF interest and importance to the fat man is F. A. Hornibrook's book, "The Culture of the Abdomen".[2] In regard to the title, it is interesting to note that one of the meanings given for the word "culture" in the Oxford Dictionary, though described as obsolete, is the training of the human body. The author shows how it is possible by care and regular exercise to prevent and even to eliminate the prominent abdomen characteristic of men of a certain type after they have passed middle age. The exercises are clearly described and are quite simple. Medical practitioners will find this a useful book to give to their patients.

[1] "A Practical Medical Dictionary", by T. L. Stedman, A.M., M.D.; Twelfth Edition; 1933. London: Baillière, Tindall and Cox. Royal 8vo., pp. 1267, with numerous illustrations. Price: 35s. net.

[2] "The Culture of the Abdomen: The Cure of Obesity and Constipation", by F. A. Hornibrook, with preface by Sir William Arbuthnot Lane; 1934. Australia: Angus and Robertson. Demy 8vo., pp. 111, with illustrations. Price: 6s. net.

The Medical Journal of Australia

SATURDAY, MAY 12, 1934.

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THE TEACHING OF ANÆSTHESIA.

One of the most important discussions that took place at the recent Hobart congress was that dealing with the teaching of anæsthesia. Its importance arose from the acknowledged inadequacy of present teaching methods and from the large part that anæsthesia takes in modern medical practice. No medical graduate can be in practice long before he is called upon to give an anæsthetic—in almost every confinement some form of anæsthesia is needed, minor surgical procedures of all sorts call for anæsthetic agents, and the young graduate is expected by older practitioners to be able to take charge of a patient while abdominal and other operations even of the greatest magnitude are undertaken. Moreover, many practitioners in the early days of practice, especially in the cities, look to anæsthetics as likely to tide them over the early lean years.

The main point at issue in the Hobart discussion, as reported in The Medical Journal of Australia of February 17, 1934, at page 250, was how far teaching should be carried in undergraduate days. Some difference of opinion was expressed in regard to the advisability of teaching students the essentials of special techniques and of gas anæsthesia. All speakers agreed that present teaching methods were inadequate, and everyone argued that post-graduate courses in anæsthesia should be held. Dr. Geoffrey Kaye, in his opening paper, drew attention to the scheme of teaching adopted at the McGill University, Montreal, and at the same time he expressed the opinion that it was unlikely that an Australian university would adopt such a comprehensive scheme. Dr. Kaye is probably right in his contention, for according to his description of the course, detailed instruction in the administration of specialized forms of anæsthesia is given at McGill. There is no need, as Dr. Kaye pointed out, to attempt to treat an undergraduate student as if he were a post-graduate student. Undoubtedly the student should receive instruction by lectures that would give him an insight into the theoretical background of the subject, and he should study in the laboratory the effects of anæsthesia on animals. This would prepare him for the taking of accurate observations on the human subject. All participants in the Hobart discussion were in agreement that post-graduate courses in anæsthesia should be held. To quote Dr. Kaye once more: "It is most desirable that a medical practitioner who wishes to learn a new technique, should be able to do so." This is a matter that should be considered as soon as possible by the Federal Council for Post-Graduate Work, if and when it is formed.

In the Hobart discussion on the teaching of anæsthesia there was one important omission. No mention was made of the need for teaching to students the technique of local and regional anæsthesia. The subject of local anæsthesia is still in its infancy; in the future it will be extensively studied. There are many reasons why this should be so. These reasons include the advantages from the patient's point of view and those claimed by surgeons; they have been described in this journal on many occasions. In a country like Australia, where medical practitioners often have to work alone at great distances from others, and when the advantages of local anæsthesia have been recognized by medical practitioners and patients, it will in large measure displace general methods. Dr. Kaye, in his paper at the congress, suggested that the subject of the teaching of anæsthetics should be

considered by the Federal Council of the British Medical Association in Australia and that the Federal Council should take the matter up with the several Australian universities. If this were done, the subject of local anæsthesia should be included in any recommendations made.

Current Comment.

FŒTAL RESPIRATION.

J. Barcroft and others have carried out extensive experimental investigations concerning the conditions of fœtal respiration.[1] It had been found in the rabbit that, as pregnancy advanced, the blood leaving the uterus and returning to the mother became increasingly dark. By the twentieth day of pregnancy the placenta and the volume of blood in the fœtal vessels attained half of their ultimate measure while the embryos were insignificant in size. Thence onward the oxygen need of the fœtus was out of proportion to the supply and the hæmoglobin of the maternal venous blood became more and more depleted of the gas, the blood in the uterine veins becoming darker and darker. The fœtus then grows in an environment the oxygen of which is ever falling, just as in the ascent of Mount Everest. The embryo receives oxygen from the blood plasma. The mountain climber gets it from the air and his acclimatization depends on factors concerned with modifications in pulmonary respiration and processes affecting blood composition. The former has no place in embryonic life. In mountain climbing hæmoglobin is increased and there is a shift in the oxygen dissociation curve in the sense of greater affinity of the blood for oxygen. Barcroft and his fellow workers used goats, all "stocked" about the same time. The period of gestation is twenty-one weeks, and the kid at birth weighs over 2,000 grammes. Observations began at the end of the seventh week, when the fœtus weighs about ten grammes. The curves of control goats not "stocked" within twenty-one weeks, though not identical, fell within a very restricted area. The dissociation curves of "stocked", but non-pregnant goats fell within the same area. One pregnant goat in the seventh week showed a dissociation curve in the same area. From the tenth week onwards the curve began to deviate to right of normal, that is, the oxygen became more easily detachable from the hæmoglobin. By the eighteenth week the 50% saturation point of the maternal curve had moved nine millimetres to the right of the mean normal.

In embryos of the ninth and tenth weeks the curve seemed to fall within the normal area. Determinations made on the weeks just subsequent gave the impression that as the maternal curve shifted to the right, the fœtal curve deviated to the left. But this was not quite borne out by the facts. In any case it is only a partial representation of them. In the fifteenth week the 50% saturation point of the fœtal blood is to the left of that in the tenth or eleventh. But here the fœtal dissociation curve differed in essential shape from that of the mother. At about 90 millimetres the two cross and thence downwards diverge. The proportionate distance between them increases progressively as saturation diminishes. At between 80% and 40% saturation the fœtal and maternal curves are roughly parallel, separated in the fourteenth week by about 13 millimetres. Later the gap is greater, the maternal curve having deviated further to the right, while the fœtal curve remains about the same as in the fourteenth week. The alteration in the position of the curve seems to be related to hydrogen ion concentration. Maternal blood, as pregnancy proceeds, loses in base; there is a progressive drop in the alkali reserve of the plasma. But alterations in hydrogen ion concentration cannot account for the peculiar features of the blood in the fœtal circulation. It is not proved that the alkali reserve of the fœtal blood is higher than in normal goats or rises as pregnancy progresses. It seems that the alkali reserve of the fœtal plasma is intermediate between that of normal goats' plasma and that of goats in later pregnancy, and it is maintained at a fairly constant level. At the end of pregnancy the fœtal contains more base than does maternal blood.

The question arises whether the fundamental characters of fœtal and maternal hæmoglobins are identical. Barcroft discusses this point at some length. He refers to experimental work by Adair and McCarthy pointing to differences; and he reviews certain indirect evidence indicating that it is not unreasonable for the two to differ. He admits that what actually takes place is obscure and he hesitates to interpret the problem of which the solution is not complete. The tension of oxygen in the blood reaching the fœtus is not higher than in that leaving the placenta for the mother. There was no convincing evidence of oxygen secretion on any great scale in the placenta. Barcroft holds it as proved regarding the mother that from the middle of pregnancy onwards the dissociation curve deviates to the right, a fact which may be explained by an increase in the hydrogen ion concentration of maternal blood. As regards the fœtus, generally from about the same period the dissociation curve of the fœtal blood, at the relevant oxygen pressures, falls to the left of the normal position. This shift is due not to abnormal alkalinity of the fœtal blood, but to a specific form of hæmoglobin. In later pregnancy fœtal blood is richer in base than is maternal. The amount of carbonic anhydrase in the fœtal blood is small in comparison with that in maternal blood, particularly in the early stages, but Barcroft finds no ready explanation of this fact.

The investigations of Barcroft and other workers are of great importance, but only touch the fringe of a vast subject. Barcroft wisely refrains from reaching dogmatic conclusions on insufficient data. Much more must be known before compre-

[1] *The Lancet*, November 4, 1933.

hensive deductions can be made regarding these processes. Even then one cannot say how far the findings in some mammals are applicable to the human species.

SPECIFIC DESENSITIZATION IN HAY FEVER.

No satisfactory explanation has ever been offered of the mechanism of specific desensitization in allergic states. A desensitization literally means a neutralization of sensitizing antibodies. Whether improvement follows as a result of a decrease in these antibodies in the tissues or is due to the production of more circulating antibodies against the antigen is quite unknown. Whatever happens, the skin sensitivity frequently remains unaltered by specific treatment and the result of such treatment is frequently unsatisfactory. David Harley, working under the direction of Dr. John Freeman at the Inoculation Department of Saint Mary's Hospital, London, reports the effect of specific treatment on the skin sensitivity and on the serum reagin (or antibodies) of ten cases of hay fever due to grasses.[1] According to the practice of this clinic, the patients were all treated by injections of gradually increasing strength of Timothy grass pollen made up with alkaline saline solution and 0·5% carbolic solution, in dilution ranging from 40 to 100,000 units per cubic centimetre. The patients had all been sufferers from hay fever for many years. They were taught to give themselves the injections daily, after receiving instruction in inoculation technique, in the estimation of dosage and in the alteration of dosage according to the previous reaction. They were each supplied with a bottle of adrenaline to control any general reaction. Beforehand and at subsequent periods the skin reactions were determined quantitatively by the "prick" method, first introduced by Sir Thomas Lewis during his work on skin reactions to histamine. This method consists in a vertical prick, one millimetre deep, by a standard hypodermic needle, through a drop of the allergen placed upon the skin. The characters of the resulting wheal are traced upon a glass slide. Each patient reached a maximum dose of pollen extract equivalent to 100,000 units by the time pollination was at its height, and a maintenance dose of 50,000 units was repeated during the season. Harley ultimately reached the following conclusions:

1. That the reagin content or sensitizing power of the serum in the untreated case is proportional to the skin sensitivity. This was estimated after intradermal reactions with graded strengths of extract had been provoked in certain areas of the skin of normal individuals under which hay fever serum had been injected.

2. That skin sensitivity is actually diminished and practically abolished by treatment. This has apparently been a constant finding in Freeman's clinic, in distinction to that of Rackemann, Cooke, Levine and Coca. Harley explains these discrepancies on the ground of the scratch or intradermal technique being inferior to the prick method and on the ground of doses larger than 5,000 units, the maximum employed in other inquiries, being necessary to modify appreciably the skin reaction.

3. That the reagin content of the serum is diminished by treatment. The same normal person was used throughout to establish passive sensitization, and it was readily shown that the reagin content of hay fever serum fell rapidly once skin sensitivity had been abolished.

The clinical results were excellent. Complete relief from hay fever was obtained in all ten patients, even under conditions conducive to heavy dissemination of pollen. Other workers have been less fortunate in their cures, but have not persisted to the degree of abolition of skin sensitivity, nor have they used the large doses so persistently advocated by Freeman and his co-workers.

Discussing the influence of these findings on the comprehension of the mechanism of specific desensitization, Harley subscribes to Dale's view that histamine or a histamine-like body is actually responsible for an allergic reaction, and he himself has shown that this substance is produced only by the interaction of antigen and reagin *in vivo*, not *in vitro*. He postulates that the allergen of the specific injection combines with the reagin of the serum, so that no free allergen gains access at once to the sensitized cells, but only very gradually in subnoxious amounts. The histamine substances, produced gradually as a result of their slow desensitization, Harley presumes to be neutralized by the adrenaline which they call forth from the adrenals. He suggests that probably the patient's tolerance to histamine is also slowly raised during this process. (Harley has shown further that one cubic centimetre of serum from a hay fever patient requires over 40 units of pollen extract for its complete neutralization in the skin of a normal person.) After a period, in the inadequately treated patient, there may be a redistribution of reagin from serum to skin and a return of skin sensitivity. Hence the necessity for pushing the dose of pollen extract well beyond that of diminution in skin sensitivity. An adequately treated patient has to manufacture a complete supply of reagin. Whether this can be done is not yet decided.

Such investigations are a revelation of the attention to detail and the ingenuity displayed by research workers in allergy today; they create a spirit of optimism in a depressing therapeutic field. The application of specific desensitization to hay fever patients has been in general the most successful of all, and the issue is less often clouded by the problem of multiple sensitivity. Whether Harley's view of the mechanism involved, which incidentally is rather reminiscent of Ehrlich's views, is accepted or not, it certainly fits the experimental and clinical facts. The technical methods employed at Saint Mary's Hospital should be carefully considered and, if possible, emulated in Australian allergy clinics.

[1] *The Lancet*, December 30, 1933.

Abstracts from Current Medical Literature.

THERAPEUTICS.

Xanthine Diuretics.

A. R. BLISS, JUNIOR, AND R. W. MORRISON (*Journal of Laboratory and Clinical Medicine*, December, 1933) report the results of their experimental studies on the diuretic properties of the following xanthine derivatives: theophylline calcium salicylate (phyllicin), theobromine calcium salicylate (theocalcine), theophylline sodium acetate ("Theocin soluble"), theobromine sodium salicylate ("Diuretin"), theophylline and theobromine. The effects of graduated doses of these drugs were noted on adult rabbits. Twenty-four-hour specimens of urine were collected and the daily observations included the total volume of urine voided, determination of the physical characteristics of the urine, the specific gravity, reaction, total solids, uric acid, urea, albumin, sugar and microscopic examinations. The results are tabulated and discussed in detail, together with a review of the literature. It is evident from the data that the xanthine derivatives used are capable of producing a good diuresis in rabbits, inducing increased elimination of solids and uric acid and maintaining a diuretic action for from two to five and a half days after the cessation of administration. It is also obvious that, in the overdosage established during the study, these agents are capable of producing gastro-enteritis, nephritis and subsequent death. If the increase in urine output, the increase in solids eliminated, safety and the duration of the diuretic action are taken into consideration, phyllicin in doses of 50 to 60 milligrammes per kilogram three times a day is most efficacious. Theophylline comes next in efficiency and then theocalcine, the dosage being the same.

Therapeutic Uses of Sodium Hyposulphite.

J. KABELIK (*La Presse Médicale*, August 23, 1933) describes treatment with sodium hyposulphite of many and various conditions. Investigation of its usefulness in anaphylactic conditions, such as eczema and asthma, in intoxication by arsenic, mercury and bismuth, led to the theory that its action was one of colloidal protection. On this assumption the author extended its application to many diverse diseases with very satisfactory results. In eclampsia, intravenous injections of hyposulphite have been used for five years at an obstetric clinic. During this period there has been no death from eclampsia, whereas formerly the average was two *per annum*. And even better results have been obtained in preeclamptic conditions, in the uncontrollable vomiting and other toxicoses of pregnancy. In extensive burns the primary toxic effects result from colloidal changes in the blood serum. In such conditions intravenous injections of hyposulphite, several times daily, have a most beneficial effect on the general condition, as well as allaying pain. It is the most efficient anti-anaphylactic and also acts as a therapeutic complement in serum, or in chemotherapy, especially where the serum or drug is ill-borne or lacks activity. Thus in some cases of rheumatism sodium salicylate has much more effect if accompanied by hyposulphite injections. In neuralgias, even in obstinate trigeminal neuralgia, striking results were obtained; 20% of patients were completely and sometimes immediately cured after one to three injections of 0·2 gramme; 50% reacted less speedily, but were improved after more prolonged treatment; 30% showed no improvement. In metallic poisoning the hyposulphite frees the metal from its combination with albumin. In three cases of lead poisoning so much lead was mobilized in this way from the deposit in the bones that acute lead intoxication was produced. In acute and chronic poisoning by alcohol and the alkaloids, by hydrocyanic acid, by lysol, and by phenol, the injection of hyposulphite rouses the patient from coma and acts favourably on any ulceration that may have resulted. The author has used hyposulphite of soda, magnesium, calcium, copper and lead. The magnesium salt is twice as potent as the sodium. It has more narcotic effect, which is useful in treatment of neuralgia, and is more effective in eclampsia. But a serious objection is that magnesium hyposulphite in large doses produces hepatic degeneration.

Treatment of Tuberculosis.

C. H. COCKE (*Annals of Internal Medicine*, December, 1933) makes a plea for conservatism as the basis of all treatment of infection by the tubercle bacillus. His thesis can be summed up—rest is the only specific. He points out that he is considering the best method of handling the private tuberculous patient and is not attempting to solve the social and economic problem of the disease. All the tried forms of therapy are summed up: climate, tuberculin, gold and sodium thiosulphate, sanatorium and rest, artificial pneumothorax, the vaccines and surgical procedures. The common factor of all these treatments, from the sanatoria to the most extensive surgical intervention, is rest. There is a tendency among many to resort to surgery earlier and earlier in the disease, until the point has been reached of assuming that surgery is the royad road to an early cure. This will never be, for the tuberculous lesion is present after the operation as well as before it, and this procedure cannot be a substitute for the healing process, but an aid thereto. The patient is quite easily led into a false sense of security and then fails to observe the fundamental rules which are essential to the healing process. This is not to say that surgical procedures have not a place in the treatment of pulmonary tuberculosis, but the fundamental principles on which all the various forms of intervention depend is rest to the diseased tissues.

Irritable Colon Treated by Sodium Ricinoleate.

GEORGE N. BURGER (*Journal of Laboratory and Clinical Medicine*, December, 1933) reports the results of treatment by capsules of sodium ricinoleate ("Soricin") of twelve patients exhibiting the symptoms generally attributed to irritable colon, namely, abdominal pain, distension, constipation, passage of excessive flatus, borborygmi, nervousness, frequent appearance of mucus in the stools, palpable and tender colon with radiographic evidence of spasm. It has been shown earlier that bacterial toxins can be neutralized by "Soricin" *in vitro*. Later it was proved that certain of the intestinal flora to which patients were highly sensitized could be similarly detoxified *in vitro* and the idea then arose that these toxins might be neutralized *in vivo*. It was also demonstrated that "Soricin" really diminished the skin sensitivity to heat-killed autogenous stool vaccine. The author set out to determine the effect of "Soricin" on the symptoms and skin sensitivity to the autogenous stool vaccine in these cases. Doses of 0·3 to 1·8 grammes (5 to 30 grains) were given four times daily, before meals and at bedtime; the course lasting two to three months. In view of the fact that all these patients had failed to respond to a bland diet and antispasmodics, the author felt certain that "Soricin" was of definite benefit in the treatment of irritable colon, for the symptoms were generally much improved. His results also confirmed the view that there was a decrease in the skin sensitivity to bacterial vaccines following this treatment

Gangrene due to Thromboangiitis Obliterans.

SAUL S. SAMUELS (*The Journal of the American Medical Association*, February 10, 1934), in reviewing twelve cases of gangrene due to *thromboangiitis obliterans*, sums up his experiences of some 300 similar cases during the last eight years. Extreme conservatism is the basis of his treatment, only one patient requiring amputation in this large number of cases. No single remedy or procedure is advocated; it is the intelligent combination of various fundamental factors that is of importance. Rest in bed in the horizontal position is the first fundamental principle. Next, stress is laid on the necessity for prohibiting smoking, a healthy granulating ulcer changing its appearance in a few hours if smoking is resumed. Thirdly, intravenous hypertonic (2% to 3%) saline solu-

tion injections are used as a mechanical aid in the enhancement of collateral circulation in the extremities. Three hundred cubic centimetres are given every other day until gangrene and ulceration are healed. In cases without ulceration clinical improvement is noted in improved nail growth, increased warmth of the extremities and cessation of intermittent claudication. Lastly, correct surgical principles must be applied in the local treatment of the ulceration and gangrene. The aim of this local treatment is to aid in the development of the line of demarcation which marks the site of spontaneous amputation of dead tissue. Foot baths of a mild antiseptic lotion are given for ten minutes or so once a day. This is followed by the liberal application of an anæsthetic ointment to relieve the pain. Only rarely is it necessary to resort temporarily to the opiates for relief. The author considers that sympathectomy and ganglionectomy have no place in the treatment of the disease.

NEUROLOGY AND PSYCHIATRY.

Amnesia in Relation to Crime.

J. S. HOPWOOD AND H. K. SNELL (*Journal of Mental Science*, January, 1933) have investigated amnesia in relation to crime in 100 male persons in the State Criminal Asylum at Broadmoor, at whose trials amnesia for their crimes was alleged. They conclude that the distinction between true and simulated amnesia is often difficult to make. Neuropathic inheritance and a history of chronic alcoholism or the possession of a psychopatients in 1926. There is, they claim, in those cases in which there was true amnesia. When true amnesia is present, the crime is frequently without apparent motive. When there is a confused recollection of acts and partial amnesia, the ideas accompanied by marked emotion are generally remembered, while those to which there is less emotional feeling attached are forgotten. The authors believe that there is nothing incompatible in the fact that statements made soon after the crime may be obliterated subsequently by a true amnesia. The loss of memory prior to the committal of the crime is usually of much shorter duration than the amnesic period following the act. Any sudden return of memory almost certainly suggests malingering.

Diabetes Insipidus.

PURCELL G. SCHUBE (*Journal of Nervous and Mental Disease*, November, 1933) endeavours to throw fresh light upon the nature and treatment of *diabetes insipidus* by means of encephalography. Though rare, this illness is known to accompany certain abnormal mental states, and the author claims that investigation along encephalographic lines has not hitherto been attempted. He chose three psychotic cases in which the symptom-complex was displayed and subjected each of the patients to a complete laboratory investigation. The X ray films generally showed little or no ventricular abnormality. The third ventricle and the fluid areas at the base of the brain were within normal limits and none of the cases showed any abnormality of the *sella turcica*. Fractional cell counts and protein determinations were made without yielding any evidence as to the pathology of the condition, and the spinal fluid chemistry findings were equally unsuggestive. It has previously been observed that a remission of the symptoms of *diabetes insipidus* occurs after lumbar puncture and the withdrawal of fluid. This author was able to confirm this, as in two of his cases the symptoms were alleviated. In the third case there occurred no change either in the *diabetes insipidus* or in the mental state, the latter being that of mental retardation. The author was therefore unable to throw any fresh light upon the pathology of *diabetes insipidus*.

Polycythæmia Vera.

N. W. WINKELMAN AND M. A. BURNS (*The Journal of Nervous and Mental Disease*, December, 1933) have studied the neuro-psychiatric features presented by two cases of *polycythæmia vera*. In addition to the plethora, splenomegaly and cyanosis which these authors observed, there were such subjective complaints as headache, dizziness and scotoma. The mental phenomena did not fit in with any psychiatric symptom-complex, but confusion and mental deterioration were prominent. Other observations in the psychiatric sphere included impairment of memory and progressive dementia. An autopsy was performed upon one case and the outstanding feature was the prominence and dilatation of the cerebral vessels both large and small. Huge venous sinuses were formed within the brain substance. Engorgement and stasis within the brain resulted in anoxæmic changes in the ganglion cells. The vessels were extremely tortuous and in places they had become denuded of their lining elements. Such a microscopic picture served to distinguish this condition from any other and to explain the neuro-psychiatric concomitants.

Brain Tumours in Psychotic Patients.

GERALD R. JAMEISON AND GEORGE W. HENRY (*The Journal of Nervous and Mental Disease*, November, 1933) present detailed investigations carried out upon psychotic patients in whom a cerebral tumour was verified by *post mortem* examination. The presence of the tumour was diagnosed in only 30% of the cases prior to death; in other cases the clinical symptoms were complicated by such factors as senility, arteriosclerosis, syphilis and alcoholism. These authors confirm the findings of Baruk, who made similar observations on forty-one patients in 1926. There is, they claim, no psychosis peculiar to cerebral tumour, but the presence of a tumour will serve to modify, according to its situation within the brain, the symptoms of whatever psychosis it happens to complicate. A notable feature of these cases is that the patient sometimes interrupts his psychotic behaviour and talk to complain that he has a headache, feels giddy or ill. In other cases, although the patient may not be able to describe his feelings accurately, the attention of the physician may be drawn to the possibility of a brain tumour being present by changing contrasts and incongruities in the symptoms and signs. There may, for instance, be periods of impulsive violence, great irritability, followed by dulness and stupor. It would appear probable, from the research presented by these authors, that the extremely irritable, anxious excitable and aggressive periods represent "distress from localized pressure or the milder degrees of increased intracranial pressure, while the periods of retardation, somnolence, confusion and memory disorders represent a degree of increased intracranial pressure which seriously interferes with all conscious activities".

Contraception and Mental Hygiene.

HANNAH M. STONE AND HENRIETTE HART (*Mental Hygiene*, July, 1933) claim that the relation of contraception to mental health is twofold: mental defects or disturbances may become aggravated by pregnancy or may be transmitted to the offspring unless some contraceptive method is utilized, and inadequate contraceptive knowledge may engender certain psychopathic conditions. An analysis of the cases treated at the Mental Health Centre in Newark showed that 71% of the women were referred to the clinic because they themselves were suffering from some psychotic or psychoneurotic condition. In the majority of instances the psychoneurosis was an anxiety state, the underlying cause of which was fear of pregnancy. In a number of instances wives had applied to the centre on account of the neurotic state of their husbands, which in the majority of cases was found to be due to improper methods of contraception, particularly *coitus interruptus*. These authors claim that their follow-up reports indicate that sound contraceptive information led to the amelioration of many of these neurotic states and that reliable contraceptive advice formed a valuable therapeutic measure in the treatment of the psychoneurosis; and further, that a wider dissemination of such advice as part of an extended mental hygiene programme would in a number of cases help to prevent the onset of those marital maladjustments which so frequently underlie the psychoneuroses.

Special Articles on Treatment.

(Contributed by request.)

XXXI.

THE TREATMENT OF MAXILLARY SINUSITIS.

THE maxillary sinuses are very prone to infection by microorganisms, and their infection may lead to inflammatory conditions of the pharynx, larynx and the bronchial tree. It may be the source of a focal infection, and also of disorders of the gastro-intestinal tract and even, in rare instances, infection of the orbit, optic nerve and meninges. For this reason it is essential that the general practitioner should be familiar with the recognition and at least the palliative treatment of this condition. In passing, the writer stresses the importance of the natural ostium of the maxillary sinus as the main port of entry of infection to these cavities. Less frequently the infection spreads from diseased conditions about the apices of teeth, and more rarely still from infected dental and dentigerous cysts. The maxillary sinuses are present in young children and may be involved in inflammatory processes which need treatment. It is most desirable that the reader should be familiar with the anatomy of the region in order to apply treatment the more successfully.

As the treatment of chronic maxillary sinusitis is essentially surgical, our attention in this article will be directed to the treatment of acute and subacute inflammation.

When a diagnosis of acute maxillary sinusitis has been made, treatment should be instituted at once in an effort to relieve it and to facilitate its resolution. Every "cold in the head" may be looked upon as a condition of more or less acute suppuration of the nasal accessory sinuses, and many "colds in the head" lead to a chronic nasal discharge. Every "cold in the head" must be looked upon as a possible forerunner of chronic nasal accessory sinusitis. Treatment should be directed to reducing the congestion of the mucous membrane in the neighbourhood of the natural ostium; for by so doing drainage may be reestablished.

Acute Sinusitis.

There are certain routine measures which may be resorted to in the treatment of acutely inflamed sinuses.

At the onset a hot bath, with the ingestion of large quantities of water may be beneficial to the patient. He should then be confined to bed and given a liquid diet. The bowels should be freely opened, preferably by means of calomel followed in seven or eight hours by a liberal dose of magnesium sulphate or such suitable saline purgative. Aspirin, "A.P.C." or Dover's powder may be administered at intervals according to requirements. Hot fomentations may be applied to the face. Smoking should be forbidden. During the stage of acute congestion it is perhaps preferable to refrain from local treatment. Irrigation of the nasal cavities with watery solutions during this stage is followed by increased turgescence of the mucosa and may lead to middle ear inflammation. Also this form of treatment is likely to remove the protection of the mucoid secretion. Steam inhalations may afford comfort and may be used in the acute stage. The vapour of compound tincture of benzoid is soothing to the mucosa and is used by adding one teaspoonful to the pint of steaming water contained in a quart jug; the fumes are inhaled for ten minutes at a time and several times a day.

At a little later stage various medicaments which produce shrinkage of the mucous membrane may be added to the steam. Menthol is a commonly used drug in this connexion, and the general practitioner is advised, if he prefers it, to use it in the form of an alcoholic solution rather than the crystals.

Such a prescription as the following is recommended.

℞

Menthol 1·2 grammes (18 grains).

Sp. Vini Rect. 15·0 cubic centimetres (four drachms).

One-half to one teaspoonful should be added to the pint of steaming water in the manner described above.

The patient should avoid cold air and draughts, especially after taking steam inhalations.

In adults, nasal drops in the form of 0·12 gramme (two grains) of menthol to 30 cubic centimetres (one ounce) of liquid paraffin or "Argyrol" (10% in glycerine), or in children 0·3 gramme (five grains) of resorcin to 30 cubic centimetres (one ounce) of olive oil, may be found helpful in relieving the nasal congestion and symptoms.

Subacute Sinusitis.

After the acute stage has passed the practitioner may temporarily shrink the swollen mucosa by the local application of a solution of cocaine. No advantage is to be gained by using a strong solution of this drug, and usually a 1% or 2% solution will produce the desired effect. Adrenaline, either alone or with cocaine, is to be avoided, as the engorgement and swelling which follow the initial shrinking may last for several days. The solution should be applied to the region of the middle turbinate bone, and under this structure, if it can be managed, by means of a fine cotton wool mop. To make the application satisfactorily, the practitioner is advised to use a nasal speculum, a head mirror and reflected light or a head lamp. If such illumination is not available, he may direct the mop to the region of the middle turbinate by feel and guesswork, or a spray of the above solution may be used in place of the direct application. Neither of these methods is as satisfactory as the application of the drug under vision. This, however, requires a certain amount of skill for its performance, and the practitioner should make himself familiar with the necessary manipulations whenever opportunity offers, in order to be competent when an occasion as the above arises.

There are drawbacks to the use of cocaine. The main one is that the relief is only temporary, since the shrunken mucosa soon relaxes and returns to its former swollen condition. It may even become worse than it was before the drug was used. However, it frequently happens that one application causes a free flow of serum or pus, or both, from the sinus, and this brings about relief from the acute symptoms.

Heat, either as hot fomentations or as dry heat from bran bags which have been heated in the oven, or derived from electric light baths, may be applied to the outside of the face with beneficial results in the early stages. Some oto-rhino-laryngologists advocate the douching of the nose with as hot a lotion as can be borne by the patient with the head held in a lower position than that of the body and tilted to the opposite side. A suitable solution is made up of one teaspoonful of salt and one of baking soda in a pint of warm boiled water; two or three Condy's crystals may be added.

Treatment by suction may be tried. A Politzer bag or Higginson's syringe or similar apparatus with an olive-shaped nose-piece attached may be used. The bulb is emptied of air by squeezing it in the hand, the nasal attachment is inserted into the nostril of the affected side, the nostrils are closed by squeezing them between the fingers, the patient is directed to swallow, and at the same time the bag is allowed to expand suddenly. A negative pressure is set up in the nostrils, and pus may be drawn from the maxillary sinus by such means. It is a method of doubtful value; but it may be worth trying, especially if there is room beneath the middle turbinate to allow a small plug to come away from the natural ostium.

As the natural ostium is situated high up on the lateral nasal wall, it is only in early stages of acute maxillary sinusitis that relief may be afforded by the methods described above. When the case has advanced to a stage of definite retention of the products of the inflammation of the sinus mucosa, that is, empyema, the practitioner must be familiar with certain surgical procedures if he

wishes to continue his attempts at further treatment. Where an oto-rhino-laryngologist is available, his services should be sought at this stage.

Failing this, the medical practitioner may puncture the lateral nasal wall under the inferior turbinate bone or through the membranous portion of the wall under cover of the middle turbinate bone, or the sinus may be intubated through the normal ostium by a specially curved cannula. Of the three the route under the inferior turbinate is recommended and is performed as detailed below.

The following apparatus is necessary: (i) a suitable trocar, such as Lichtwitz's, or a straight trocar and cannula; (ii) a Higginson's syringe with a rubber attachment to fit the large end of the trocar; (iii) warm, sterile, normal saline solution, a solution of boracic or even of such antiseptic as cyanide of mercury (1 in 8,000), contained in a suitable bowl; (iv) a large bowl to catch the solution escaping from the nose during lavage.

The medical practitioner is advised to sit or stand in front of the patient, and by means of a nasal speculum and a head light, or head mirror and reflected light, to inspect the lower part of the nasal chamber. He should gently swab the mucosa of the floor and of the inferior turbinate along its free edge with a cotton wool mop which has been dipped into a 10% solution of cocaine, to which a few drops of adrenaline solution (1 in 1,000) have been added. After a few minutes' interval the mucosa in the region treated will be found to have shrunken and to have become ischæmic. Attention should then be paid to the area under cover of the inferior turbinate bone, the mop being advanced without causing undue pain to the patient. A mop moistened with cocaine solution can be left in this position, or a strip of gauze (a width of half an inch is recommended) soaked in the cocaine solution and squeezed out, may be inserted beneath the inferior turbinate bone and packed there. No advantage is gained by keeping the mop or gauze tape in position for longer than ten minutes.

Before inserting the trocar it is suggested that the practitioner "try out" the anæsthetic by pressing the mop hard against the lateral wall. If no discomfort is occasioned by this manipulation he can then proceed to puncture the wall. He stands in front and to the right of the patient. The straight trocar, with its cannula, is held with its base in the palm of the right hand and with the shaft between the tips of the thumb and first and second fingers. Guided by illumination, the point is inserted under the inferior turbinate bone to the distance of 2·5 centimetres (one inch) or so. The hand is then moved inwards to a more mesial position and dropped somewhat, the point being thereby directed backwards, upwards and outwards. It is then advanced in this direction, and it should impinge on the lateral nasal wall at its thinnest part, near the attachment of the inferior turbinate bone. The patient's head should be steadied during the manipulation by the operator's encircling and holding it close to his body with his left arm. The trocar is advanced gently, but firmly, the operator exerting only a little pressure, until the crunch of the breaking down of the bony partition is felt, and it is then advanced a little further to make sure that it is within the cavity. The point of puncture is about 2·5 centimetres (one inch) from the end of the nose. If resistance is encountered the trocar point is probably in too low or too anterior a position and is in contact with dense bone. It should be withdrawn and another place selected. After the wall is punctured care should be exercised, the point must not be advanced too far, and no attempt should be made to push it on against resistance; for at times, in certain abnormalities, the postero-external wall particularly may be punctured, and the point would then lie beyond the sinus. Should such an accident happen, the subsequent injection of air or lotion would cause an immediate swelling of the face and marked pain. This accident happens quite frequently in the hands of the inexperienced, and the learner must be watchful. If the point of the cannula is pointed too high, the roof of the antrum may be punctured and severe and alarming symptoms may develop, particularly if air or lotion is injected.

When it is felt that the point is inside the sinus, the trocar is withdrawn, leaving the cannula in place. This is now connected to the syringe. Some experts advise against the injection of air; but the writer prefers to "try out" by gently squeezing air from the syringe. If the air passes through easily (which is manifest by the bubbling of the air escaping from the ostium, indicating that there is a free passage for any fluid which may be introduced) the lotion is then injected (very gently at first, until the flow is established) and is caught in a basin held under the chin by the patient. Serum and pus contained in the sinus will be washed out with the lotion. The lavage can be finalized by again blowing air through, which expels the remaining lotion from the sinus. Sometimes it is impossible to obtain a free flow of air or lotion. The practitioner is cautioned against using force, as serious damage may be done thereby. In such an instance the normal opening is probably blocked by a swollen piece of mucous membrane (polypus) lying over the opening, or the point may actually lie between the bony and mucosal walls of the sinus. This latter is more likely to occur when there is an acute exacerbation of infection in a case of chronic sinusitis; in such a case the mucous membrane may be fibrous and tough, and it may then be pushed ahead of the trocar point. If the operator is sure that the point of the cannula is lying within the sinus, and obstruction is present, he must desist, and he may now try the effect of suction. This may be done by detaching the rubber tube connexion from the cannula, firmly squeezing the bulb, reattaching the rubber tube to the cannula, and allowing the bulb to relax. By this means pus may be sucked from the sinus.

Following either procedure the cannula is withdrawn. Next day lavage by air and lotion may be easily performed. Sometimes in early stages of acute retention of pus in the sinus (empyema) the condition subsides with one washing. This is especially so when the retained fluid is serum, the presence of which is manifested by the appearance of a straw-coloured thin fluid either with displacement or with suction. In most cases it may be necessary to repeat the performance daily for several days before the cavity becomes clean.

The operator may carry out a suction aspiration by quickly injecting a small amount of lotion into the antrum from an ordinary syringe and then quickly sucking it back again.

Should lavage fail after several attempts to clear up the condition, the next stage in treatment is to make a large opening into the sinus. This should be done by a nasal surgeon, but should the practitioner not be in a position to call upon the services of an expert and wish to perform this operation, he, after cocainizing the part, may enlarge the opening under the inferior turbinate by using a suitable rasp or punch forceps. Since a description of this operation is beyond the scope of this paper, the practitioner is referred to text books on the subject for information.

The presence of an outward pressure of the middle turbinate prevents in most instances the proper ventilation of the sinus and so hinders the resolution of the inflamed mucosa. It may be advisable to remove the anterior half or so of this bone. This can be done, after thoroughly cocainizing the region, by the use of middle turbinate scissors and snare or by a conchotome inserted laterally with one blade medial and the other lateral to the bone. Drainage via an inferior antrostomy is always more efficient when a partial middle turbinectomy has been done.

Should the discharge fail to clear up, the practitioner must keep in mind the fact that diseased conditions of teeth may be the cause, and attention should be directed to them.

The writer warns the practitioner against the drawing of healthy teeth to provide a means of drilling the alveolus and draining the antrum this way. The method served its usefulness at a time when knowledge of maxillary sinus disease had not reached its present state. It is obsolete and should not now be employed.

The use of autogenous vaccines is said by some to be effective in cleaning up sinusitis after drainage has been established; but the writer cannot recommend the method.

On the other hand, intramuscular injections of "Collosol" manganese in doses of one to two cubic centimetres at intervals of two or three days sometimes acts in a remarkable way, provided drainage has been established.

Acute and subacute sinusitis must be carefully controlled and treated until cleared. If this is done, many cases of chronic sinusitis can be prevented.

Chronic Sinusitis.

The treatment of chronic sinusitis is essentially operative. It is outside the scope of this paper and therefore will not be discussed.

Discussion.

In the summing up of this discussion it is as well to point out that the primary cause of infection of the maxillary sinus is swelling of the mucosa around the natural opening and subsequent blocking and retention of the products of inflammation of the sinus mucosa. Almost invariably a pressure of the middle turbinate in an outward direction is found as a predisposing cause of "colds in the head", and swelling of the nasal and sinus mucosa is an accompaniment of "colds in the head".

In children the occurrence of sinusitis is more prevalent than is recognized and is invariably associated with diseased tonsils and adenoids. Removal of these diseased structures brings about a shrinking of the swollen nasal and sinus mucosa, and in favourable cases sinusitis may be cured by this means alone. Furthermore, the presence of adenoids in the child interferes with the passage of air through the nose, and as the development of the nose (width in particular) depends in a large measure upon this factor, it is essential that diseased adenoid tissue should be treated or removed as soon as it is discovered. By this means the development of middle turbinate obstruction, which blocks the natural opening into the maxillary sinus, would most likely be prevented.

The writer feels that many instances of diseases of the nasal accessory sinuses, and of the maxillary antrum in particular, could be prevented if diseased adenoid tissues were removed earlier, and even when middle turbinate obstruction had already developed, if attention were paid to this abnormality.

Deficiency in certain essentials in diet is considered by some authorities to be an important factor in the production of adenoids and nasal accessory sinus disease. Therefore, good food, including vitamins such as are contained in cod liver oil, fresh fruit juice, "Ostelin", "Irradol A", and the like, is indicated after diseased tonsils and adenoids have been removed.

The practitioner will find useful reference and guidance in the following two books by British authors: "Diseases of the Nose, Throat and Ear", by Logan Turner and others, and "Diseases of the Nose and Throat", by Sir St. Clair Thomson, a new edition of which is about to appear.

R. GRAHAM BROWN, F.R.A.C.S.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the University of Adelaide on November 30, 1933.

The Interpretation of Systolic Murmurs.

DR. GUY A. LENDON read a paper entitled: "The Interpretation of Systolic Murmurs" (see page 613).

DR. E. BRITTEN JONES congratulated Dr. Lendon on his paper. He disagreed with the statement that rheumatic arthritis was rarely seen at the present time. He had been struck by the number of patients suffering from acute rheumatism that had been admitted to the Children's Hospital in the past two or three years.

With regard to Professor Cleland's *post mortem* results, in which rheumatic disease of the aortic valve was found in a few cases in which the mitral valve was apparently normal, Dr. Britten Jones thought it extremely rare for rheumatism to attack the aortic valve unless the mitral valve was affected too.

With regard to the greater frequency of mitral stenosis (with presystolic bruit *et cetera*) in females, which had been mentioned by Dr. De Crespigny, experience with rheumatic children had shown him that in most cases the rheumatic girl with endocarditis left hospital with a systolic bruit and an early diastolic bruit, which signs he interpreted as being the earliest signs of stenosis, that would lead subsequently to the development of a presystolic bruit; whereas the rheumatic boy whose heart was affected generally left hospital with a harsh systolic bruit, the diastolic element being absent.

Epithelioma of the Lip.

SIR HENRY NEWLAND showed a man who had had an extensive epithelioma of the lip in the left angle of the mouth removed several years previously. Sir Henry Newland had repaired the resulting facial defect by a pedicle graft brought across from the left fronto-temporal region.

Diathermy of the Prostate.

DR. I. B. JOSE showed a man, aged fifty-five years, who had had attacks of acute retention of urine for two years and had been considered then to be suffering from a urethral stricture. He had had a small fibrous prostate, observed cystoscopically. Under a spinal anæsthesia the obstruction had been resected *per urethram*, with a Canny Ryall resectoscope. He had now no residual urine and his micturition was free and normal. Dr. Jose said he had shown this patient to illustrate a notable advance in prostatic surgery in the treatment of this type of prostatic obstruction. This had been made possible by the mechanical development of the operating endoscopic instruments and the improvement in diathermy machines, providing an instrument that could cut under water and also give a coagulating current when required to deal with bleeding points.

He stated that the types of cases in which this method of treatment had been found to be of the greatest use were those in which prostatectomy might be contraindicated owing to the type of prostatic disease, cardiovascular disease, renal disorder, or senility.

A MEETING OF THE OBSTETRICAL AND GYNÆCOLOGICAL SECTION of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, Albert Street, East Melbourne, on March 13, 1934.

Puerperal and Non-Puerperal Pelvic Inflammation.

DR. ARTHUR SHERWIN read a paper entitled: "The Ætiology and Treatment of Puerperal and Non-Puerperal Pelvic Inflammation" (see page 617).

DR. G. B. BEARHAM said that it gave him great pleasure to discuss Dr. Sherwin's most interesting, well planned and thoughtful paper.

He contented himself with a discussion on the pathology of the acute and chronic salpingitis as raised by Dr. Sherwin. One seldom had to report on the pathology of specimens of acute salpingitis, as the practice at the Women's Hospital was to treat expectantly all such cases, whether Neisserian, post-abortal or puerperal in origin. He did not like to express an opinion as to the ætiology of a specimen of an acutely inflamed tube after inspection macroscopically and microscopically, unless he had especially stained for organisms *in situ* by Gram's method *et cetera*. On recognizing the infecting organism, one could then class the case as Neisserian, puerperal *et cetera*.

In his experience the appearance of acute salpingitis was the same, whether gonococcal, streptococcal or staphylococcal in origin. Macroscopically the tube was

reddened, inflamed, and almost turgid with its epithelium, sticky and covered with serous or mucopurulent material, or just œdematous, with a droplet of pus exuding from its fimbrial end. Nearly always there was an associated inflammation of the proximal portion of the broad ligament.

Microscopically one noticed the folds of the lining of the tube pressed and crowded together with a varying degree of destruction of the columnar epithelium and a massing of small round cells beneath the epithelium, together with an engorgement of the blood vessels. This latter appearance Dr. Sherwin referred to as perisalpingitis.

Acute salpingitis either completely resolved, excepting for a deposition of connective tissue to a varying degree beneath the epithelium, or gave rise to a general peritonitis, or passed through a subacute to a chronic stage. If it did not resolve, the pus exuding from the fimbrial end soiled the peritoneal cavity, giving rise, particularly in the streptococcal and mixed *Bacillus welchii* cases, to a general peritonitis with usually a fatal termination. The pus exuding from the fimbrial end might set up an inflammatory reaction, causing a gluing of the fimbria to the ovary, bowel, posterior layer of the broad ligament or its fellow of the opposite side. This sealing of the tube then gave rise to an increased collection of muco-purulent material within it, resulting in the characteristic retort tubes which lay in the pouch of Douglas and which were commonly associated with the gonococcus, but which might follow the streptococcus, enterococcus *et cetera* of a less than ordinary virulence. The purulent or mucopurulent contents of the retort tubes then slowly changed, so that the inner walls became covered with a cheesy material and the cavity filled with a thin watery coloured fluid. This corresponded to the subacute salpingitis. Later the cheesy material disappeared, giving rise to the condition of chronic hydrosalpinx which later might become reinfected from organisms in the gut being liberated by some intraabdominal crisis or by a lowering of the patient's general resistance. The organisms travelled along the adhesions or per medium of the lymphatics or by way of the blood stream to the contents of the tube, giving rise to a recurrent pyosalpingitis.

One might get a sudden hæmorrhage into a hydrosalpinx, giving rise to the condition called hæmatosalpinx; this condition clinically simulated a twisted ovarian cyst or an ectopic gestation.

In some cases, instead of the above process, the perisalpingitis gave rise to multiple small abscesses beneath the columnar epithelium; usually here the organism was the staphylococcus. These small abscesses might burst into the lumen of the tube, giving rise to a pyosalpinx or peritonitis, or might subside, giving rise to a chronically thickened, somewhat nodular tube with, in some cases, a patent lumen. This condition sometimes resembled tuberculous salpingitis, which usually reached the pathologist as a member of the chronic group of salpingitis.

The commonest infecting organisms were the gonococcus, streptococcus, staphylococcus and *Bacillus welchii* associated with *Streptococcus fæcalis* or *Bacillus coli communis*.

Where it was a reinfection of a chronic salpingitis the most common organism was the *Bacillus coli communis* or enterococcus. The mode of entry of the organisms in acute salpingitis was invariably by way of the uterine mucosa to the columnar epithelium of the tube, where the infection was Neisserian.

In the case of the other organisms, they might follow the same route as the gonococcus, as Dr. Bearham had been able to satisfy himself when doing *post mortem* examinations on puerperal cases, that the infection had travelled directly from the uterine cavity through the uterine end of the tube into the tube itself.

However, more commonly the route of infection was either through the lymphatics to the broad ligament and thence to the tube or per medium of the sinuses of the placental site to the veins of the broad ligament or to the ovarian veins and thence to the tube by means of an infective thrombosis, or per medium of the blood stream as a septicæmia or pyæmia—the former usually streptococcal and the latter usually staphylococcal.

In some cases the peritoneum was infected first, as in a perforated uterus, where sepsis had supervened and the tube became involved secondarily in the general peritonitis. Less frequently salpingitis followed an extrauterine primary source of infection, as in the case of subacute bacterial endocarditis *et cetera*.

Tuberculous salpingitis, in Dr. Bearham's experience, was very rare, as in eight years he could remember seeing only two specimens, which would make its incidence less than quoted by Dr. Sherwin.

DR. FELIX MEYER said that the systematic treatment of pelvic inflammation at the Women's Hospital, Melbourne, dated practically from the early nineties. Before that time, during his four years of house-surgeonship, intraabdominal pelvic surgery at the hospital had been, generally speaking, restricted to ovariotomy (ovarian parovarian cystomata) and hysterectomy (serrenœud and clamp).

On one occasion only did he see the pelvis drained *per vaginam*, and that was when, as house surgeon, having diagnosed a pelvic abscess, he asked and got permission to push a trocar into Douglas's pouch, following it up with an incision and draining free pus.

In the early nineties the conservative surgery of tubes and ovaries found its first advocacy in Martin, of Berlin, and Pozzi, of Paris, but Dr. Meyer's colleagues of the new gynæcological staff at the Women's Hospital were not at first greatly impressed by this new idea. (Speaking personally, after hearing Pozzi's paper at the British Medical Association meeting in England and visiting his clinic in Paris, Dr. Meyer stated, he reacted to his main principles of conservatism.)

For some years it had been a recognized principle at the Women's Hospital to open the abdomen for acute conditions, such as pyosalpinx, and to remove tube or tubes inflamed or bulging with pus or adherent to the deep pelvis. With a tubo-ovarian abscess the ovary was always removed, and if there was a doubt as to the condition of the other ovary, it was also removed. He recalled the friendly shakings of the head by way of comment on his first resection of an ovary with salpingostomy.

By far the greatest number of cases of pyosalpinx in patients admitted to the Women's Hospital, Melbourne, were of gonorrhœal origin, and at first in nearly every case a double salpingectomy was performed. As time went on they realized the natural history of tubal inflammation, the meaning of hydrosalpinx and the lessons indicated, and he was safe in saying that for some of them on the gynæcological side of the hospital expectant treatment took the place of operating in the acute stage of salpingitis, with results incomparably better than the tragic happenings under the old methods.

For some ten years before the termination of his connexion with the Women's Hospital (1918) Dr. Meyer rarely opened the abdomen for acute tubal conditions (tubal gestation, of course, excepted). Posterior colpotomy was the rule. All his colleagues, Dr. O'Sullivan, Dr. Rothwell Adam and Dr. F. W. W. Morton, favoured this procedure. Often the diagnosis between the acute appendix and salpingitis was difficult. In those cases laparotomy was always done. Looking back, with the true perspective of experience, he realized the overwhelming advantages of the expectant method over the immediate in the surgery of clearly defined acute pelvic inflammation.

PROFESSOR R. MARSHALL ALLAN congratulated Dr. Sherwin on his clear presentation of the difference in pathology and sequelæ between gonorrhœal and puerperal infections of the pelvic tissues. He was pleased with the reference to the work of Curtis, of Chicago, who was the main protagonist in America of conservative treatment of such conditions. To him they owed much for the demonstration of the short life of the gonococcus in the tubes, while streptococcal infections remained virulent for a long period of time. With the increase in the prevalence of abortion, much of it septic in origin, pelvic infection from this

source had relatively increased. One point of considerable importance was that such streptococcal infections tended to subside gradually and subsequent sterility was less frequent than after a gonococcal infection of equal severity. Similarly, one should remember that many mild cases of gonorrhœa tended to recover, leaving the tubes patent.

Dr. Sherwin was right in emphasizing non-interference during the acute stage of pelvic infections. Early operation could result only in radical removal of organs with consequent sterility. The only indication for operation was the drainage of abscess formations. General measures for the improvement in health, together with the local application of heat by medical diathermy, douches and packs, should be persevered with, and no operation should be considered in streptococcal cases for at least two years because of the high rate of mortality and morbidity associated with such procedures. He pointed out the value of estimation of the blood sedimentation rate in deciding when to operate in such cases. Occasionally the abdomen might be opened by mistake, and although it took some courage to do so, the operator should immediately close it and resort to conservative methods. The results of such action were surprisingly good. Finally, he stressed the importance of prophylaxis, which could be summed up as gentleness in all manipulations and operations, care with the delivery in all cases, and a recognition of the harmfulness of repeated examinations in obstetric work.

DR. A. E. COATES thanked Dr. Sherwin for his comprehensive ar interesting paper. He appreciated the compliment of being invited to speak at a sectional meeting of which he was not a member.

As a general surgeon he met with cases at times which presented difficulty in the diagnosis. A condition of pelvic peritonitis was present, but whether tubal or appendiceal in origin might be hard to determine. In most cases there was, of course, no difficulty, but in an odd case he had found a gangrenous appendix *plus* salpingitis. In three cases he could recall yellow vaginal discharge and yet the condition was appendiceal. In two cases the gangrenous appendix was discharging down the uterine tube. The absence of any digestive trouble and the incidence of pain and discomfort in the pelvic organs made a diagnosis of acute salpingitis easy. There were, however, patients admitted to general hospitals, sent in by reputable practitioners, labelled with a diagnosis of appendicitis, and even in the presence of doubt in the mind of the surgeon he felt that it was wiser to open the abdomen. If an acutely inflamed appendix were left by mistake, the results might be more disastrous than the opening of the abdomen in an acute salpingitis. Further, when a case was obviously one of acute salpingitis, opened possibly under the impression that it was a case of appendicitis, the problem of what to do with the offending organs was a real one. He agreed that in mild tubal leakage the pus was not harmful and the abdomen could be closed with a drain tube inserted.

But what of the more severe types with fairly extensive subomental peritonitis? He felt that at any rate in older women no great harm could come from the removal of such tubes at once. There was plenty of authority for such a procedure in the writings and lectures of eminent surgeons who had spoken from this platform of recent years. He had learnt by experience to diagnose these cases with greater accuracy and confessed to opening fewer of them than in earlier years of practice, but still there were perhaps two a year which were opened by mistake, and in these he removed the tubes if a severe peritoneal infection were present in older women, whereas in younger women he used drainage.

He would profit by the discussion, and doubtless would be even less radical in the future. The peritoneal adhesions formed after tubal and uterine infection were apparently less troublesome as causes of intestinal obstruction than were adhesions about the ileo-cæcal area after appendicitis. The ileo-cæcal area was very vulnerable from the point of view of obstruction, and the pus of the *Bacillus coli communis* infection might possibly be more likely to produce such adhesions.

Dr. Coates had observed that secondary metastases from septic uterine and tubal conditions were usually mild. If abscesses developed in the course of a pyæmia from pelvic sepsis (apart from gonorrhœa), they were usually readily cured by incision. Even pyogenic collections in the knee joint and the skull bones from such a source were cleared up by incision with comparatively little loss of function.

Dr. Coates thanked the various contributors to the discussion for the clear statement of their views on a subject which, for him, had been surrounded by a good deal of doubt and misgiving.

DR. W. H. CUSCADEN agreed with the conservative attitude of the paper.

The Fowler position, diathermy and shock therapy in more chronic cases gave the best results. Not enough attention was given to persevering with the treatment of pelvic infections. The patient was put out of hospital too soon after the temperature fell. With perseverance 80% of the cases of pelvic infection would clear up on conservative treatment.

Removal of one tube was useless, as nearly 40% required subsequent operation. The removal of both tubes was not always satisfactory; in many cases hysterectomy was indicated and the slightly greater risk was more than balanced by the better end-result.

In reply to a question of Dr. Coates, Dr. Cuscaden said it was sometimes necessary to open the abdomen in cases in which it was impossible to decide whether the infection was appendiceal or tubal in origin. In these cases, if the condition was definitely a salpingitis, the abdomen should be closed without anything further being done. With definite general peritonitis, drainage through stab wounds was indicated. It was interesting to note that the greatest drainage was on the left side, indicating, apparently, extension along the left paracolic gutter.

Dr. Sherwin, in reply, thanked those present for their attendance, and the speakers for their interesting contribution to the evening, especially Dr. Meyer, for the trouble he had taken in refreshing his memory by looking through the hospital records made during his first association with that institution. He agreed with others speakers that, should an abdomen be opened under the suspicion that pathological conditions other than acute salpingitis were present and should the latter only be found, the correct procedure was to close the abdomen without interference with the affected tubes.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Saint Vincent's Hospital, Darlinghurst, on September 21, 1933. The meeting took the form of a series of demonstrations by members of the honorary staff.

Fibromyoma of the Stomach.

DR. H. H. BULLMORE showed a married woman, aged thirty-two years, who was admitted to hospital on December 30, 1932, complaining of loss of weight during the previous six months. More recently she had had anorexia and vomiting.

On examination slight resistance was present in the epigastrium. Succussion was present. Nothing of importance was detected otherwise.

On January 16, 1933, a barium meal showed that the stomach and duodenum were apparently normal. On January 20, 1933, a barium enema revealed no abnormality. On January 27, 1933, a fractional test meal revealed anacidity.

On February 3, 1933, a barium meal revealed a constant irregularity of the lesser curvature suggesting early adenocarcinoma. On February 10, 1933, gastrectomy and gastroenterostomy were performed by Dr. Coppleson. The patient's subsequent history was uneventful.

The pathological report of the specimen consisted of a stomach tumour and gland from the lesser curvature. Macroscopically there was a moderately hard, hemispherical, solid tumour projecting into the lumen of the stomach in the pyloric region. The mucosa had been

pushed aside apparently by this mass and formed a thick circular collar around its base. The cut surface of the tumour showed what appeared to be fibrous tissue in whorled arrangement. Sections examined microscopically showed a fibromyoma (fibroid) growing from the muscular coat of the stomach. The free surface was covered by a layer of granulation tissue rich in blood vessels and so hæmorrhagic. The whole texture of the fibroid was opened out by acute inflammatory œdema. Very numerous pus cells appeared in the interstices of the fibromyomatous tissue. The mucosa of the stomach showed an acute inflammatory infiltration and was swollen. The mass was held to be benign, as spindle cell sarcoma could be eliminated.

Dr. Bullmore pointed out that these tumours were rare and references in the literature were scanty. The lymph glands examined showed only very brisk inflammatory hyperplasia; the germ centres were increased in size and activity. No sign of any malignant metastasis was apparent.

Werlhof's Disease.

Dr. Bullmore also showed a woman, twenty-one years of age, who was admitted to hospital on June 8, 1932, complaining of intermittent attacks of pain in the vagina with, at the same time, inability to pass urine, of four years' duration. Hæmaturia and ecchymoses were of three months' duration. Since admission and up to August 10, when splenectomy was performed, she had very frequent attacks of epistaxis, bleeding from tongue and gums; purpuric spots appeared on the tongue and palate and mucous membrane of the cheeks. Coarse purpuric rashes appeared on many parts of the body, and large and small ecchymoses on the limbs, as well as hæmaturia.

Cystoscopic examination revealed oozing of blood from the anterior wall of the bladder. Gynæcological examination failed to reveal any abnormality.

The urine, with the exception of blood, was normal. Blood examination revealed a moderate degree of secondary anæmia, with a normal white cell count, but the platelets were below 1,000 per cubic millimetre. Coagulation time was about normal, and bleeding time was increased to thirteen minutes. Blood calcium was 15 milligrammes per 100 cubic centimetres. The thrombocytes on June 27 were not sufficiently numerous to estimate.

Adrenaline was injected at times during epistaxis; it had temporary effect. Protein shock in the form of muscular injection of milk at first seemed to stimulate the formation of platelets, as the count mounted to 4,000 and a few giant thrombocytes were seen in the blood smears. The improvement was only temporary.

On August 3, 1932, a blood transfusion did much more harm than good. Very large ecchymoses appeared and the epistaxis was very severe. The red cell count fell to such an extent that splenectomy was advised. A transfusion with 800 cubic centimetres of citrated blood was carried out before operation.

Sir John McKelvey operated on August 10, 1932. The thrombocyte count at the time was below one thousand. On August 12 it was 30,600, on August 15 it was 190,000, and on August 23 it was 350,000. The count then fell to 90,000 on September 5, but again rose to 360,000 on September 13. Blood estimations were done regularly until January of this year, when the platelet count was 220,000. No further count was made until the fifteenth of the present month, when the platelet count was 150,000. The patient had not had any bleeding since the operation.

Neoplasm of the Lung.

DR. O. A. A. DIETHELM showed a labourer, aged forty-six years, who complained of cough with a good deal of expectoration and some dyspnœa. He was admitted to hospital on April 14, 1933.

In October, 1932, he had an hæmoptysis and again in March, 1933, and during the interval he had had several feverish attacks, often preceded by shivering feelings. With these he used to perspire profusely and he coughed up large quantities of sputum. In April, 1933, he had severe pain in the lower part of the left side of the chest anteriorly. He had not lost any weight.

On examination he had signs of thickened pleura and in the upper part of the left side of the chest anteriorly and posteriorly dulness with diminished breath sounds and resonance, the former being absent over small areas. There was complete dulness anteriorly on the left side, more marked than in any other part of the chest, extending from the second to the sixth space and coincident with the cardiac dulness. In the axilla, just below the axillary space, there was dulness with some bronchial breathing suggesting some fibrosis. The heart was markedly displaced to the left side, the apex beat being in the axillary line.

There was no history of venereal disease and his sputum contained no tubercle bacilli. The blood did not react to the Wassermann test; the test was done a second time, with the same result. X ray examination of the chest on April 13, 1933, showed the whole of the left side of the chest to be dull throughout. The heart and mediastinum were displaced into the left hemithorax. The appearance suggested gross pleural thickening or possibly massive collapse of the lung. The right lung appeared clear. The Casoni and hydatid precipitin tests gave no reaction. The blood count was normal; there was an eosinophilia of 3%.

X ray examination was repeated on April 21, 1933, and showed no alteration. The report stated that the pleural thickening was too dense for the underlying structures to be seen. X ray examination after injection of lipiodol on May 3, 1933, revealed complete obstruction of the left main bronchus from the bifurcation of the trachea, suggesting an endothelial neoplasm with obstruction and atelectasis of the lung. Bronchoscopic examination revealed a fleshy growth blocking the left bronchus twelve inches from the mouth.

The patient was then given a course of deep X ray therapy of six treatments from June 6, 1933, to June 16, 1933, and a radiogram taken on June 17, 1933 showed some expansion taking place in the lung—in the apex, periphery and costo-phrenic angle. The trachea was not so much displaced to the left as on the previous examination. The remainder of the lung field was obscured owing to atelectasis.

The patient left hospital and reported on August 7, when his symptoms and signs were less pronounced, while another radiogram revealed almost a normal chest. The dulness on the left side had almost completely cleared. Evidently it was due to atelectasis and the bronchus had now cleared. The heart and mediastinum had retracted practically to a normal position. There was still some dulness to the left of the heart shadow and some enlargement of the hilar nodes.

In view of this remarkable improvement another Wassermann test was done on August 10, 1933, with negative results. The patient went home, but had to be readmitted on September 7 with a history that he had an attack of influenza on August 21, accompanied by severe paroxysmal coughing with expectoration of muco-pus and great dyspnœa, especially on the slightest exertion. No hæmoptysis occurred. He complained also of a sense of constriction on the left side and had had some shivering feelings and sweats. He had lost some weight, but not a great deal. His temperature on admission was 38·3° C. (101° F.), and he had been running an intermittent temperature. The sputum again contained no tubercle bacilli. He had a leucocytosis of 14,800. The Wassermann test gave an incomplete negative result. X ray examination of the chest revealed complete dulness through the whole of the left lung field with deviation of the trachea to the left. The appearance indicated massive consolidation, most probably due to neoplasm.

Probably, on account of the remarkable effect of the irradiation, the primary condition was a bronchial carcinoma (medullary) similar to the oat-celled sarcoma of Barnard. Unfortunately it was not possible to get a small piece of the growth for biopsy when bronchoscopy was carried out, as although Dr. Marsh could get a pair of forceps down to the growth, when he attempted to take a piece, it bled so much that he did not care to persist.

Dr. Diethelm thought, from clinical examination after relapse of the condition subsequent to his remarkable improvement after irradiation, that, although the patient might have a recrudescence and extension of the growth, his signs could more easily be accounted for by an unresolved pneumonia supervening. The signs, particularly in the bronchial character of the breath sounds, suggested this. Posteriorly also, below the scapula in the paravertebral region, there were the signs of a bronchiectatic cavity. It was possible, of course, that the origin of the condition, especially with such a long history and very little loss of weight, might be syphilitic, but there was no venereal history or any treatment in the past to suggest any venereal history, while his response to the Wassermann test was negative on three separate occasions, although it was an "incomplete negative" on the fourth. The Wassermann test would be repeated and a Kahn test would be carried out as well. It had not been considered necessary before. Subsequent treatment would depend on progress, but if no contraindication arose further irradiation would be given.

Epigastric Tumour.

DR. J. E. SHERWOOD showed a female patient, aged thirty-eight years, who was in good health until three years ago, when she commenced to suffer from nausea after food. Four months previous to attending hospital she noticed two small "lumps" appearing on the front of the upper part of the chest. These lumps persisted and increased in size. She also suffered from pain extending down the inner side of the right arm. She had a son, eighteen years of age, who was in good health.

On the occasion of Dr. Sherwood's first examination three months before: (i) there was a fairly firm epigastric tumour present, not tender, moving freely with respiration, probably continuous with the liver; (ii) there were two rather cystic swellings, one on each side, extending downwards and outwards from the regions of the sterno-clavicular joints. There were no other markedly abnormal features on examination. Both pupils reacted to light and accommodation and were equal in size. Both knee jerks were present, though sluggish.

On June 13, 1933, the blood Wassermann test gave a "+++" response. A blood count revealed the following:

Red cells, per cubic millimetre	4,200,000
Hæmoglobin value	70%
Colour index	0·83
Leucocytes, per cubic millimetre	9,800
Neutrophile cells	73%
Eosinophile cells	Nil
Lymphocytes	23%
Monocytes	3%
Basophile cells	1%

Slight anisocytosis and poikilocytosis were present. No macrocytes or nucleated red cells were seen.

The Casoni test and the precipitin test for hydatid disease gave no reaction. X ray examination of the stomach and duodenum revealed a very low atonic stomach; no organic lesion was detected. X ray examination of the clavicles and chest wall did not reveal any abnormality. The patient was afebrile during her six weeks' stay in hospital. Antisyphilitic treatment was resorted to.

At the time of the meeting, after three months of somewhat intermittent treatment, the swelling on the left side of the chest had disappeared. The corresponding swelling on the right side was much smaller and softer. The epigastric tumour had diminished in size considerably; it appeared softer, and was also losing its outline. It seemed that the antisyphilitic treatment should effect its purpose.

Familial Dystrophy.

Dr. Sherwood also showed a female patient, aged thirty-eight years, who was a member of a family extensively affected with the complaint. In her case the complaint affected both hands and forearms, muscular wasting and weakness being very evident. Both feet and legs were affected similarly and the lumbar musculature had not escaped. The appearance of the calf muscles was better than their capabilities.

The patient walked on a broad base, and to rise from a horizontal posture had to climb up on herself in the usual typical manner. Dr. Sherwood showed a family tree which was based only on the statement of members of the family. It showed that three generations were affected.

Paget's Disease.

Dr. Sherwood's next patient was a male, aged sixty-nine years, who stated that two years previously he had to obtain a hat two sizes larger than his usual one. Four months ago he had a "sudden" syncopal attack. His previous health had been "satisfactory". During his stay in hospital he had slight hæmorrhage *per rectum*.

On examination prominent tortuous vessels, especially on the forehead, were visible. A large cranium was in evidence. The chest appeared fixed and emphysematous. He appeared somewhat pot-bellied. The pelvis was broad and splayed out. He was also bow-legged. X ray examination of the skull revealed well marked Paget's disease. The same condition was present in the pelvis. X ray examination of the rectum and colon showed a condition of diverticulitis. The blood did not react to the Wassermann test.

Myelomatosis.

In showing the next patient Dr. Sherwood said that he was entirely indebted to the Coast Hospital for the diagnosis. Owing to the rarity of the condition he felt that the case should be demonstrated.

The patient, a male, aged thirty-four years, had complained of a constant feeling of tiredness over a period of two years, noticeable chiefly when resting, less marked when working. He had always had some nasal obstruction as long as he could remember.

He had been married for six years and had one child, three years of age. Both wife and child appeared healthy.

Twelve months previously he had been confined to his bed for a period of three weeks with pleurisy and pneumonia. Some months later, for a period of ten days, he was said to be suffering from lobar pneumonia. Five months ago he complained of stabbing pain in the left side of his chest, which was then diagnosed as pleurisy.

Three months ago Dr. Sherwood referred him for admission to the Coast Hospital, where his condition was diagnosed as myelomatosis. During his stay in hospital he evidently had very profuse epistaxis. Shortly after leaving that hospital he suffered from a spontaneous fracture of the neck of the left humerus.

Throughout life he seemed to have had repeated attacks of epistaxis. At three years of age he developed scarlet fever. At seventeen yars of age he had adenoids removed with apparent severe post-operative hæmorrhage. Six months ago he had had his antrum punctured.

On examination very marked general cachexia and pallor were present. Septic teeth and unhealthy tonsils were visible. No abnormalities were detected in the abdomen or chest. Apart from a systolic murmur over the pulmonary area and a friction rub, also systolic in time, possibly pericardial, over the same area, no abnormalities could be found on examination of the cardio-vascular system. The pupils were equal and reacted to light and accommodation. The knee jerks were active. There was no tenderness to pressure over bones in any situation.

Urine examination revealed a trace of Bence-Jones protein. The blood did not react to the Wassermann test.

A blood count revealed the following information:

Red cells, per cubic millimetre	2,190,000
Hæmoglobin value	50%
Colour index	1·19
Leucocytes, per cubic millimetre	9,400
Polymorphonuclear cells	55%
Large lymphocytes	5%
Small lymphocytes	35%
Eosinophile cells	1·5%
Large mononuclear cells	1·5%
Fragile forms	1·5%
Turk irritation cells	0·5%

The red cells showed slight anisocytosis and very slight poikilocytosis. No nucleated red cells were seen.

On X ray examination of the bones myelomatosis was reported.

Dr. Sherwood, quoting from Nelson's "Loose Leaf Surgery", said that myelomata were a group of tumours derived from the cells of bone marrow, in some respects resembling sarcomata, but differing in histogenesis. The tumours were usually multiple, appearing simultaneously in many bones, as reddish or greyish masses, generally limited to the skeletal system, possibly due to a general affection of the bone marrow. The ribs and sternum were the most frequent sites affected. The vertebræ, skull, femur, pelvis and humerus, in the order named, were less frequently involved. Spontaneous fractures were frequent; bending of bone might occur from deficiency of lime salts. Metastases in other organs were rare. The growths varied in size from a bean to an orange. In all types Bence-Jones protein appeared in the urine (as in leuchæmia *et cetera*).

It was a disease of middle life, occurring chiefly in males from twenty-five to seventy years of age. It might be infectious in origin and ran a relatively slow course. Microscopically the tumours might consist of any one of five varieties of cells: myelocytes, myeloblasts, lymphocytes, nucleated red cells, plasma cells. The most frequent type was constituted of plasma cells.

Hey Groves and Griffiths, writing in the "Medical Annual" of 1932, stated that all forms of treatment had been hitherto considered futile, and it was interesting to read an article by W. B. Coley, of New York, on the use of the mixed toxins of erysipelas and *Bacillus prodigiosus*, together with the effects of radiation, in the amelioration of this condition, the treatment in some cases causing disappearance of the tumours. Coley quoted one patient alive and well for five years after a course of the mixed toxins had been given twice a week for two years. Apparently this was the only case on record of multiple myeloma in which recovery under any form of treatment had occurred.

Acromegaly.

Dr. Sherwood's last patient was a male, aged sixty-five years, who had been well until two years previously, when he commenced to suffer from headache, occipital in situation. The pain, "throbbing in character", had persisted off and on ever since.

On examination a receding forehead was noticeable. The face appeared elongated from vertex to chin. The zygomatic areas seemed unduly prominent; the jaw was enlarged and undershot. The jaw enlargement seemed to have progressed markedly during the last two years, as also the zygomatic prominences. The limb changes were not noticeable.

X ray examination of the pituitary fossa two years before revealed no abnormality. More recent X ray examination disclosed a little calcification in the pituitary gland; no abnormality of the fossa was detected.

Blood pressure readings were slightly on the low side, the systolic and diastolic pressures being 138 and 80 millimetres of mercury respectively. The blood did not react to the Wassermann test. Apart from slight deafness, cranial nerve involvement appeared to be absent.

(To be continued.)

MEDICO-POLITICAL.

AN ORDINARY MEETING OF THE TASMANIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Tasmanian Museum, Hobart, on April 10, 1934, DR. W. E. L. H. CROWTHER, the President, in the chair.

Anti-Cancer Campaign.

The Honorary Secretary, Dr. J. H. B. Walch, read a motion that had been referred from the annual meeting of the Branch, to the effect that a Tasmanian anti-cancer campaign should be undertaken and that the Branch should do all in its power to help in the matter. It was pointed out that the Northern Division of the Branch had some time ago instituted such a campaign in northern Tasmania and had collected a large, though insufficient, sum of money for the purchase and installation of a deep therapy X ray plant at Launceston. It was now thought, however, that the campaign should be extended to embrace the whole State and that an attempt should be made in southern Tasmania to obtain the additional funds needed for the Launceston plant, which should serve the whole island. After some discussion Dr. F. W. Fay moved:

> That a committee of the British Medical Association approach the Lord Mayor and ask him to call a public meeting to extend the activity of the Anti-Cancer Campaign to the whole of Tasmania.

Dr. T. Giblin seconded the motion, which was carried.

Dr. W. E. L. Crowther, Dr. C. N. Atkins, Dr. F. W. Fay, Dr. R. B. McIntosh, and Dr. E. A. Rogers were appointed a committee to carry out the terms of the resolution, to interview influential citizens and generally to pave the way for a successful campaign and, by means of articles in the daily Press and so forth, to arrange for the education of the public in regard to cancer.

Boat Racing for Boys.

The Honorary Secretary reported that the Headmasters' and Headmistresses' Association of the Tasmanian Secondary Schools had asked the Council for an authoritative opinion as to the advisability or otherwise of boys under seventeen years of age engaging in boat racing.

The Council had been under the impression that the Council of the Victorian Branch had discussed the matter, and had asked the latter for their conclusions in regard to it. A reply had, however, been received that the discussion in Victoria had been confined to the public Press. The general feeling of the meeting was that such contests were not harmful to boys who before commencing training had been found by a medical man to be physically sound. Dr. F. W. Fay and Dr. A. W. Shugg were requested to draw up a report on the subject and present it at the next meeting of the Branch.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Barry, John Patrick, M.B., B.S., 1930 (Univ. Sydney), 6, Church Street, Randwick.

Wyndham, Norman Richard, M.B., B.S., 1931 (Univ. Sydney), 27, Oxford Road, Strathfield.

Kelleher, Frederick, M.B., B.S., 1930 (Univ. Sydney), Vanderberg's Hotel, Forbes.

Sillar, Donald Boyd, M.B., B.S., 1929 (Univ. Sydney), Alexandra Street, Kurri Kurri.

Medical Societies.

THE MEDICAL DEFENCE SOCIETY OF QUEENSLAND.

THE ANNUAL MEETING OF THE MEDICAL DEFENCE SOCIETY OF QUEENSLAND was held at the B.M.A. Building, 35, Adelaide Street, Brisbane, on February 15, 1934, DR. ALEX MARKS, the President, in the chair.

Annual Report of the Council and Financial Statement.

The annual report of the Council and the financial statement and balance sheet for the year ended December 31, 1933, were presented and adopted on the motion of the President, seconded by Dr. A. G. Anderson.

The annual report is as follows:

The Council has pleasure in submitting the following report of the Society for the year ended December 31, 1933.

Membership.

The membership of the Society is 377. During the year eleven new members were elected and one member was reinstated. Since last year our losses have been as follows: Five members have left the State, fourteen have defaulted in payment of their subscription for two years, two have resigned, and one deceased. This makes a total loss of ten members.

As reported at the last annual meeting, the death of one of our oldest members, Dr. Wilton Love, took place in January, 1933.

Office-Bearers and Council Elected for 1933.

President: Dr. A. H. Marks.
Vice-President: Dr. D. A. Cameron.
Honorary Secretary: Dr. Neville G. Sutton.
Honorary Treasurer: Dr. R. G. Quinn.
Councillors: Dr. A. G. Anderson, Dr. A. B. Carvosso, Dr. G. W. Macartney, Dr. S. F. McDonald, Dr. W. N. Robertson, Dr. Kerr Scott, Dr. H. S. Waters.

Medico-Legal.

Eleven cases were submitted to the Council during the year, four of which were referred to the solicitors for legal advice. Three of the latter have been finalized and one is still pending. The Council has dealt with the remaining cases and rendered all the assistance possible.

Amendment of Articles of Association and By-Laws.

The Memorandum and Articles of Association and By-Laws of the Society are at present in the hands of the solicitors for amendment.

Finance.

The total assets of the Society amount to £3,826 15s. 11d., of which £2,882 3s. 6d. is invested in Australian consolidated Treasury bonds and £509 7s. 6d. in Australian consolidated inscribed stock. The surplus income over expenditure amounted to £242 18s. The total expenditure amounted to £118 0s. 7d., of which £60 5s. 2d. was paid for legal expenses. The amount received for entrance fees and annual subscriptions was £211 11s. 6d. The total amount of income from investments was £143 3s.

(*Signed*) A. H. MARKS,
President.
NEVILLE G. SUTTON,
Honorary Secretary.

Election of Office-Bearers.

Dr. A. G. Anderson, Dr. Kerr Scott, and Dr. Neville G. Sutton, who retired in conformity with the By-Laws of the Society, were reelected as members of the Council for the ensuing year.

Mr. R. G. Groom, Chartered Accountant (Aust.), was elected auditor.

Proposal Regarding Life Membership.

A motion, notice of which was received from Dr. D. P. O'Brien, Rockhampton, moved *pro forma* by Dr. Neville G. Sutton and seconded by Dr. R. G. Quinn, was put to the meeting and lost. The motion was to the effect that members of the Society who had paid their subscription for twenty years should become life members without further payment of subscription.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on April 9, 1934.

The following degree was conferred *in absentia*:

Master of Surgery (*Ch.M.*): Samuel Pearlman, M.B.

A gift of £48 was received from the University Undergraduates' Association for the purchase of books for the Fisher Library.

The trustees of the estate of the late Dr. Gordon Craig forwarded a cheque for £5,936 18s., representing the balance of the bequest to the University by the late Dr. Craig for the endowment of the Department of Urology.

Messrs. Davis Gelatine (Australia), Limited, offered to provide the salary of a research worker in bacteriology in the Department of Agriculture for the years 1934 and 1935. The offer was accepted with grateful thanks.

A further gift of valuable books and journals for the Department of Surgery was received from Dr. F. P. Sandes, Macquarie Street, Sydney, and accepted with grateful thanks.

The resignation of Dr. P. C. Charlton of the Lectureship in Materia Medica was accepted with regret.

The following appointments were approved: Dr. F. C. Hardwick as Lecturer in Materia Medica in the Faculty of Dentistry; Miss Joyce Vickery, M.Sc., as Demonstrator in Botany; Dr. S. H. Lovell as part-time Demonstrator in Anatomy for Lent and Trinity Terms, 1934; Dr. M. C. Lidwill as Lecturer in Clinical Medicine at the Royal Prince Alfred Hospital; Dr. J. K. Maddox and Dr. W. A. Bye as Tutors in Medicine at the Royal Prince Alfred Hospital; Mr. F. P. J. Dwyer, M.Sc., as half-time Demonstrator in Chemistry; Dr. M. R. Flynn as Acting Tutor in Surgery at the Royal Prince Alfred Hospital; Dr. J. S. McMahon as Acting Tutor in Surgery at Saint Vincent's Hospital; Dr. A. M. Welsh as part-time Research Worker in Pathology in the Department of Cancer Research; Dr. A. J. Canny as Lecturer in Medicine.

The Honourable Sir William Cullen, K.C.M.G., M.A., LL.D., was unanimously reelected Chancellor of the University for the ensuing three years.

Sir Mungo MacCallum, K.C.M.G., M.A., LL.D., D.Litt., was unanimously reelected Deputy Chancellor of the University for the year 1934-1935.

The James King of Irrawang Scholarship was awarded to Mr. N. A. Burges, M.Sc. Mr. Burges graduated Bachelor of Science in 1930 with First Class Honours in Botany, and was admitted to the degree of Master of Science in 1932. He proposes to undertake a further course of study in mycology in the University of Cambridge. Mr. Burges has also been recommended to the Royal Commissioners of the Exhibition of 1851 as worthy of the award of a Science Research Scholarship.

On the recommendation of the Faculty of Agriculture, Mr. E. G. Pont, B.Sc.Agr., was reappointed as Walter and Eliza Hall Research Fellow in Agriculture for a further period of one year. Mr. Pont proposes to continue his research work at the National Institute of Dairy Research, Reading, England, and other similar institutions abroad.

Correspondence.

TWIN BIRTHS.

SIR: In THE MEDICAL JOURNAL OF AUSTRALIA of March 31, 1934, Dr. C. T. Underwood reported the case of a woman giving birth to twins, one a boy weighing nine and a half pounds, and the other a girl of nine pounds, and asked if any other practitioner had had a similar experience.

On April 15, 1925, I confined a little woman, two *para*, without much trouble, of twins, boys, each weighing nine pounds. The first was born at 10.25 p.m., a "P.O.P.", an easy forceps delivery, without any tearing. The second infant was a footling presentation, and was born at 10.50 p.m.

At 11.10 p.m. a large single placenta with two cords and one sac was naturally expressed.

Yours, etc.,
W. M. A. FLETCHER.

Haberfield,
New South Wales,
April 23, 1934.

DEATH FROM ANAPHYLAXIS.

SIR: In THE MEDICAL JOURNAL OF AUSTRALIA of March 31, 1934, Dr. J. G. Sleeman reports a fatal case of anaphylaxis following an injection of antitetanic serum.

I saw a case in France in 1918, in which a child was dead in less than five minutes after an injection of antitetanic serum.

A French boy of about five years of age had been knocked down on a road near the No. 2 Australian Casualty Clearing Station, where he was taken for treatment. He had lacerations and abrasions and was ordered some antitetanic serum by another medical officer. I happened to go into the dressing tent just as the serum was given. The mother picked up the child and went outside, then rushed back exclaiming "*Enfant mort!*" and he was dead.

It seemed as if the mode of death had been syncope. Unfortunately no *post mortem* was done.

Yours, etc.,
W. M. A. FLETCHER.

Haberfield,
New South Wales,
April 23, 1934.

OCULTISTS AND OPTOMETRISTS.

SIR: In reference to a letter appearing in today's issue of THE MEDICAL JOURNAL OF AUSTRALIA, under the above heading, I think it surprising that the Editor considered such a diatribe even of passing interest to the readers of our journal, but having printed it, one must conclude that correspondence of this type and tone is deemed worthy of encouragement by a journal whose primary object is considered to be the publication of matters of medical or scientific interest.

As regards the subject matter of the letter, what is not plain abuse consists of assertions, probably untrue in the main, but designed to furnish proofs for the intemperate opinions expressed by an infallible gentleman himself sitting in final judgement.

No doubt our publication has provided the desired semblance of truth which would have been lacking had the letter appeared where it rightly belongs, that is, adorning the pages of a paper serving the interests of the gentleman concerned, who so ably illustrates the maxim: "Having no case, abuse the other side."

Yours, etc.,
F. ABBEY WIESENER.

143, Macquarie Street,
Sydney,
April 28, 1934.

Obituary.

PERCY OSWALD LORD.

DR. PERCY OSWALD LORD, who died in Melbourne on March 2, 1934, was born at Dunkeld in September, 1879. He was the son of John A. Lord, an officer of the Education Department of Victoria. He went to school at Grenville College, Ballarat. After leaving school, Percy Oswald Lord undertook the study of pharmacy and in due course qualified as a pharmacist. In 1908 he decided to study medicine and became an undergraduate at the University of Melbourne. He obtained the degrees of Bachelor of Medicine and Bachelor of Surgery in 1913. After graduation he went to Sydney and was attached to the Department of Health during the smallpox epidemic. Later on he returned to Footscray, where he soon built up a large practice. During and after the war he was a quarantine officer at Williamstown. His work told on his health and in 1927 he had to give up his practice. After resting for twelve months he began practice in Collins Street. He devoted his attention to venereal diseases. He was a medical officer at the departmental venereal diseases clinic and also served on the staff of the Melbourne Hospital.

FREDERICK WILLIAM LANGTON.

WE regret to announce the death of Dr. Frederick William Langton, which occurred on May 3, 1934, at Centennial Park, New South Wales.

DAVID EARDLEY FENWICK.

WE regret to announce the death of Dr. David Eardley Fenwick, which occurred on May 4, 1934, at Wellington, New Zealand.

WILLIAM ARTHUR BOWMAN.

WE regret to announce the death of Dr. William Arthur Bowman, which occurred on May 6, 1934, at Brighton, Victoria.

ALEXANDER JARVIE HOOD.

WE regret to announce the death of Sir Alexander Jarvie Hood, which occurred on May 8, 1934, at Sydney, New South Wales.

Analytical Department.

"MILO."

"MILO" is described by its manufacturers as a fortified tonic food. They state that it contains milk and malted cereals fortified by the addition of natural vitamin extracts, organic phosphates and mineral salts (including calcium, magnesium, sodium, potassium and iron). It is claimed that vitamins *A* and *D* are present in their natural ratio, the concentrate being derived from selected fish liver oils. Care is taken to insure that the vitamins of the original grain and milk are retained; the incorporation of yeast provides vitamins B_1 and B_2. The composition of the food is stated by the manufacturers to be:

Fat	9·6%
Carbohydrates	68·1%
Ash	5·1%
Proteins	13·8%
Starch	Nil
Water	3·0%

A sample of "Milo" was submitted to our analysts, who report the following results:

Water	3·0%
Protein (N × 6·75)	14·8%
Fat	10·1%
Ash	4·6%
Nitrogen-free extract	67·5%

The presence of the following constituents was established by qualitative examination: calcium, magnesium, sodium, potassium, iron, fluorine, iodine. It will be seen that the results of the examination by our analysts are substantially in accord with the claims made by the manufacturers. "Milo" may therefore be regarded as a food suitable for use in many conditions, particularly in convalescence, general debility, insomnia and so forth. "Milo" is manufactured by Nestlé and Anglo-Swiss Condensed Milk Company (Australasia), Limited.

NOTICE.

AUSTRALIAN ARMY MEDICAL CORPS DINNER.

THE Committee of the Officers' Mess of the Australian Army Medical Corps, Second Military District, has arranged a special dinner, to be held at the Refectory of the Sydney University Union on Saturday, May 19, 1934, at 7 p.m. This dinner will be a formal function, to which will be invited the State Governor, the Director-General of Medical Services, and the senior staff officers of the Army, Navy and Air Force in New South Wales. Further, the date has been specially selected as it approximates to Empire Day and the opening of the post-graduate course arranged by the Permanent Post-Graduate Committee of New South Wales.

In view of the distinguished guests and the nature of this dinner, the Committee sincerely hopes that every member of the Australian Army Medical Corps and the Royal Army Medical Corps, whether on the active or reserve list, will make every endeavour to be present and to insure the success of the occasion. The subscription to this dinner will be approximately fifteen shillings, and this will include tobacco and wine. All those who intend to be present are asked to inform the Honorary Secretary, c.o. Victoria Barracks, Sydney, as soon as possible. Dress will be full mess dress, evening dress with miniatures, dinner jackets.

Books Received.

MEN AGAINST WOMEN: A STUDY OF SEXUAL RELATIONS, by T. Besterman; 1934. London: Methuen and Company, Limited. Crown 8vo., pp. 248, with two illustrations. Price: 6s. net.

THE CHANCES OF MORBID INHERITANCE, edited by C. P. Blacker, M.A., M.D., M.R.C.P.; 1934. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 462, with illustrations. Price: 15s. net.

CLINICAL STUDIES ON THE PHYSIOLOGY OF THE EYE, by J. G. Byrne, M.A., M.D.; 1934. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 154, with illustrations. Price: 10s. 6d. net.

Diary for the Month.

MAY 15.—New South Wales Branch, B.M.A.: Ethics Committee.
MAY 15.—Tasmanian Branch, B.M.A.: Council.
MAY 16.—Western Australian Branch, B.M.A.: Branch.
MAY 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 23.—Victorian Branch, B.M.A.: Council.
MAY 24.—New South Wales Branch, B.M.A.: Clinical Meeting.
MAY 25.—Queensland Branch, B.M.A.: Council.
MAY 31.—South Australian Branch, B.M.A.: Branch.
MAY 31.—New South Wales Branch, B.M.A.: Branch.
JUNE 1.—Queensland Branch, B.M.A.: Branch: Bancroft Memorial Lecture.
JUNE 5.—Tasmanian Branch, B.M.A.: Council.
JUNE 6.—Western Australian Branch, B.M.A.: Council.
JUNE 6.—Victorian Branch, B.M.A.: Branch.
JUNE 7.—South Australian Branch, B.M.A.: Council.
JUNE 8.—Queensland Branch, B.M.A.: Council.
JUNE 12.—Tasmanian Branch, B.M.A.: Branch.
JUNE 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

PARRAMATTA DISTRICT HOSPITAL, PARRAMATTA, NEW SOUTH WALES: Junior Resident Medical Officer.

ROYAL HOSPITAL FOR WOMEN, PADDINGTON, SYDNEY, NEW SOUTH WALES: Medical Officers.

ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Honorary Assistant Ophthalmic Surgeon.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 895, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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